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CONTINUED MORBIDITY AFTER MITRAL VALVE SURGERY

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There is today fairly general agreement on the indications for surgery in mitral stenosis and results that have much in common have been obtained in most centres. Approximately 70% of patients obtain a good result, and in the great majority of these the benefit obtained is lasting. What of the 30% in whom there is an immediate poor result or who actually regress after initial improvement? In view of the large number of patients who have undergone mitral valvotomy, the absolute number of those with poor results is not insignificant. It is the purpose of this report to discuss some of the problems presented by such patients.

Material

In April 1956, a clinic was opened under the aegis of the Cardiac Clinic at the Johannesburg General Hospital for the purpose of following up patients who had undergone mitral valvotomy. In spite of efforts to see all such patients, it soon became obvious that the great majority of the patients attending the clinic were in fact those with continuing ill-health. Very few of those with good results were seen and we were not able to trace such cases, not having the help of a social worker.

Thus, of over 300 patients subjected to mitral-valve surgery, we were personally able to follow up only 98, of whom 42 (43%) had good results, 16 (16%) fair results and 40 (41%) poor results. The result in each case was assessed by the effect of the operation on disability as graded by Wood,[1] as follows: An improvement by 2 grades or more constituted a good result, by 1 grade a fair result, and no change or deterioration, a poor result. A proportion of 'good result' cases attending the clinic were troubled by temporary complications, and they often ceased attending when the complications abated. Figures for all our cases can therefore not be given. However, the problems presented by patients who continued to attend could be divided into the following clear-cut groups:

1. 'Post-commissurotomy syndrome'.
2. Inadequate enlargement of the mitral valve orifice.
3. Late re-stenosis of the mitral valve.
4. Repeat mitral valvotomy.
5. Persistent right heart failure and/or tricuspid incompetence.
6. Combined mitral stenosis and mitral incompetence.
7. A miscellaneous group with poor results despite adequate operative enlargement of the mitral valve.

'Post-Commissurotomy Syndrome'

Recurrent bouts of this syndrome, as described by Papp and Zion[2], troubled some of our patients, irrespective of whether the functional result of surgery was good or poor. The clinical picture consists of recurrent bouts of fever with chest pain and, in some, evidence of pericarditis and left pleural effusion. Each episode is rapidly aborted by steroid therapy, the pyrexia settling within 2 or 3 days.

Case 1

A 50-year-old male, had a successful mitral valvotomy in April 1956. From 1 month after operation, he experienced recurrent bouts of substernal pain, aggravated by respiration and associated with fever. Each bout of illness was rapidly relieved by prednisone, 5 mg. q.i.d., and, in the intervals, he was asymptomatic, with a normal effort tolerance. These attacks occurred at intervals of 2-4 months, the most recent being in August 1957, 16 months after operation.

Inadequate Enlargement of the Mitral Valve Orifice

It is the aim of the thoracic surgeons operating on cases from the clinic to split stenosed mitral valves to the valve ring, or as near thereto as possible. In their earlier experience, the surgeons were often content with much less extensive splits and, even at the present time, technical difficulties occasionally preclude an adequate split. While it cannot be denied that there are instances of continued good results among subjects in whom the split was considered adequate, it is clear that a number of our patients with persistent disability were in fact those with inadequate splits. It may well be that these latter cases have re-stenosed soon after surgery, as suggested by Wood[1] (case 3). However, it must be remembered that the surgeon's description of the size of the valve orifice after surgery is only a rough indication of the residual degree of obstruction to blood flow, and the actual obstruction may be significantly different in cases with apparently equal 'long diameters' of valve orifice as described by the surgeon. Late re-stenosis may occur with a recurrence of symptoms after improvement lasting 1-2 years (case 5).

In assessing these subjects, we have appreciated that the patients' description of improvement in the first 6-9 post-operative months may be due to causes other than those

associated with the relief of obstruction at the mitral valve. It is common experience that patients may mention improvement even when only cardiotomy without valvotomy has been performed. This improvement may be psychogenic or, more important, may be due to the fact that in many subjects the operation has allowed time for intensive cardiac and respiratory medical therapy. Respiratory therapy consists of breathing exercises, postural drainage, etc., which may benefit those with chronic bronchitis, a not infrequent complication. In these subjects improvement is usually temporary and not noted for longer than 6-9 months.

Case 2

A 30-year-old female was subjected to cardiotomy in May 1954. Owing to the elastic quality of the valve, the surgeon was unable to achieve a split, but he stretched the valve orifice from a size admitting the finger to the base of the nail, to one admitting the finger to the proximal interphalangeal joint. For 6 months after operation she felt much better, her effort tolerance improving from Grade IIb to Grade I[3]. Thereafter, she rapidly deteriorated to her pre-operative status and she has experienced gradually increasing disability since then.

Late Re-Stenosis of the Mitral Valve

The question of early re-stenosis has already been mentioned. Late re-stenosis in subjects who have had *adequate* valvotomy is rare and, in our experience, is usually attributable to recurrent rheumatic activity. It is clear that rheumatic activity may occur in adults many years after the last recognised attack of rheumatic fever. Such rheumatic activity may manifest itself either by arthralgia without fever, or by overt acute rheumatic fever. As the cardiac penalty for rheumatic activity may be high and as such activity is largely preventable, it is our present routine to place all subjects undergoing valvotomy on permanent penicillin prophylaxis, either in the oral form (Phenoxymethylpenicillin—penicillin V), 120 to 240 mg. daily or Benzathine penicillin 1,200,000 U. intramuscularly once a month. In those subjects sensitive to penicillin, we have used sulphonamides and occasionally broad spectrum antibiotics.

In some of these patients with late re-stenosis, the symptoms have recommenced soon after the rheumatic activity. By contrast, symptoms attributable to mitral stenosis commence only several years after an initial attack of rheumatic fever. The development of tight stenosis in a previously normal valve would appear to be a slower process than in a previously structurally abnormal valve. However, tight stenosis may develop fairly rapidly in both groups, but the development of symptoms due to the stenosis may be related both to the degree of elevation of left atrial pressure and to its *duration*.

Case 3

A 17-year-old female with Grade II disability[4] underwent mitral valvotomy on 4 September 1953. The valve was tightly stenosed and a moderately good split to a diameter of 1 inch was performed. Thereafter she felt normal and returned to full activity. She was not on any anti-rheumatism prophylaxis. About 1 year after the operation she began to experience generalized joint pains, but did not consult a doctor. A few months later, effort dyspnoea recommenced and steadily progressed, culminating in an attack of acute pulmonary oedema during the 3rd month of a pregnancy, nearly 4 years after operation. On clinical examination, there were all the signs of tight mitral stenosis with radiological and electrocardiographic evidence of marked right ventricular and left atrial enlargement. These signs had been present before the operation, but owing to the patient's failure to attend for follow-up it is not known whether they changed during the early post-operative course. Pregnancy was terminated and repeat valvotomy advised. At operation a tight stenosis was found, with firm adhesion of the commissures, which were cut with a knife and split to the valve ring. A moderate degree of mitral incompetence was produced.

Case 4

A 27-year-old female was first seen in 1953, when she gave an 8-year history of dyspnoea, which had progressed to Grade III.[4] Clinically, there was evidence of tight mitral stenosis with electrocardiographic and radiological features of left atrial and right ventricular enlargement. At operation on 14 September 1953, a tight stenosis was found and a 'moderately good' split was performed. The excised left atrial appendage was found, on histological examination, to contain Aschoff nodes. She received oral penicillin for only 6 weeks post-operatively and remained extremely well for 2 years. Thereafter, she was assessed as having mild rheumatic activity in December 1955, and during 1956 breathlessness gradually returned and steadily progressed to Grade III disability. She experienced paroxysmal atrial fibrillation. Her electrocardiogram (Fig. 1), which after the first operation had shown marked diminution of right ventricular hypertrophy, now showed a return to the pre-operative picture, and her heart had enlarged slightly. Re-stenosis was diagnosed and repeat valvotomy advised. At operation a tight stenosis was found and digital splitting could not be performed. The anterolateral commissure was cut with a knife, and the final diameter of the mitral valve was 1½ inches.

Repeat Mitral Valvotomy

Patients of the previous 2 groups may be subjected to repeat valvotomy. This may be technically more difficult than the initial operation, partly because of more difficult entry into the previously opened left atrium. We feel that results of repeat mitral valvotomy should be considered separately from the results of the initial valvotomy. Our results in patients subjected to 2nd and 3rd operations will be presented in a separate publication.

Case 5

A 36-year-old female was first seen in 1951 with a history of progressive dyspnoea for 5 years. In May 1950 she coughed up small amounts of blood on 3 occasions. Orthopnoea and paroxysmal attacks of palpitations had been present for one year. Her disability when seen was Grade III.[4] All the clinical features of tight mitral stenosis were present. The electrocradiogram showed marked right ventricular hypertrophy and the radiological features of left atrial, pulmonary artery and right ventricular enlargement were present. The cardio-thoracic ratio was 45% and septal lines were present at the lung bases. One month before her first cardiotomy, congestive cardiac failure appeared. On 11 May 1951 cardiac catheterization revealed a pulmonary artery pressure of 92/48 mm. Hg. Pulmonary capillary pressure was not obtainable. Mitral valvotomy was attempted on 26 May 1951. The mitral orifice measured 1/8th inch in diameter with tough thickened edges. A split of the lateral commissure was obtained with difficulty. The medial commissure was partially cut with a Bailey knife. The final valve opening was 5/8ths inch in diameter. She improved considerably for 1½ years and cardiac catheterization 10 months after surgery revealed a pulmonary artery pressure of 40/18 mm. Hg and a pulmonary capillary mean pressure at rest of 12 mm. Hg. In January 1953 dyspnoea reappeared and by May 1954 she was as incapacitated as she had been before her first operation. An attempt at repeat cardiac catheterization was unsuccessful and on 20 August 1954 a second thoracotomy was performed. The mitral valve orifice was ¼ inch in diameter with considerably deformed and thick tough fibrous edges. The appendage tore during attempts at valvotomy, she bled profusely, and surgery was abandoned. Over the past 3 years she has become more incapacitated with severe dyspnoea on the slightest exertion, mild congestive cardiac failure, angina pectoris and permanent atrial fibrillation. The physical signs are unchanged. The electrocardiogram shows an increase of right ventricular hypertrophy and the X-ray findings are unchanged. Valvotomy is in our opinion still indicated in this patient and the use of newer techniques, with possible open cardiotomy, will be necessary.

Persistent Right Heart Failure and/or Tricuspid Incompetence

An interesting finding in 8 patients has been the persistence of right heart failure and/or tricuspid incompetence in

patients who, after a satisfactory valvotomy, have experienced considerable improvement in exercise tolerance. It seems reasonable to assume that after valvotomy the pressure in the pulmonary venous circulation decreases, with consequent diminished turgidity of the lungs and relief of dyspnoea. The cause of the persistence of signs of right heart failure is not clear and detailed haemodynamic investigation of these patients is planned with a view to elucidating the problem. The possibilities which suggest themselves are:

1. *Persistence of increased pulmonary arterial resistance.* It has been established[3] that in a proportion of patients who have had a successful valvotomy the pulmonary arterial resistance does not fall, but it is not clear from the reports whether such patients are symptomatically improved. Of the 8 patients we have seen with persistent right heart failure and/or tricuspid incompetence, 6 had definite electrocardiographic evidence of right ventricular hypertrophy pre-operatively and in 4 this diminished or disappeared. Thus it would seem that in at least some of these cases persistence of the increased pulmonary arterial resistance is not responsible for the clinical state.

2. *Irreversible myocardial damage.* In recent years, a great deal of attention has been focused on the mechanical obstruction to the flow of blood in patients with rheumatic heart disease. However, it is well to remember that earlier observers[4] often stressed the importance of myocardial damage in heart failure. Whether such damage may or may not be irreversible in some cases, is not known. It may be of significance that 4 of the 8 patients had moderately enlarged hearts (cardiothoracic ratio greater than 55% pre-operatively) with further enlargement post-operatively.

3. *Organic tricuspid disease* probably explains some of these cases, for it is accepted that the proportion of cases of tricuspid disease diagnosed clinically falls short of that found at necropsy.[7] Whatever the explanation, these patients continue to require medical therapy for control of their right heart failure. How their ultimate prognosis will compare with those with good results from valvotomy is not known, although it would seem that it should be significantly poorer. It is of interest that of our 8 patients, 2 had their operations more than 3 years ago, and they continue to remain subjectively well.

Case 6

A 30-year-old female was first seen in 1953, with dyspnoea Grade III-IV[8] and orthopnoea, but no paroxysmal nocturnal dyspnoea. Reversible congestive cardiac failure with evidence of tricuspid incompetence had been present off and on for 1 year. There was clinical evidence of tight mitral stenosis with atrial fibrillation. The electrocardiogram (Fig. 2) showed right ventricular hypertrophy and radiology revealed a large heart (C.T.R. 60%) with enlargement of right ventricle, pulmonary artery and both atria, and evidence of pulmonary hypertension (Fig. 3). At operation in July 1953, the valve was found to have a diameter of ⅜ths inch and was split to over 1 inch. Since operation the dyspnoea is Grade I and there is no orthopnoea. Tricuspid incompetence persists and she requires diuretics to keep her free from oedema. The electrocardiogram (Fig. 2) now shows absence of right ventricular hypertrophy and the radiological appearances are unchanged except for further cardiac enlargement (C.T.R. 64%).

Combined Mitral Stenosis and Mitral Incompetence

We are in general agreement with Belcher[5] that good results are obtained by valvotomy in a small number of patients with stenosis plus incompetence of greater than 'insignificant degree'. Nevertheless, the proportion of good results falls short of that obtained in patients without incompetence and, even though the surgeon has reported 'no increase of regurgitation' at operation, the clinical course may continue steadily downhill. It would seem that these patients are following the natural history of mitral incompetence and that their stenosis has not been a factor in causing symptoms.

Case 7

A 22-year-old female gave a history of breathlessness progressing over several years to Grade III[6] with associated orthop-

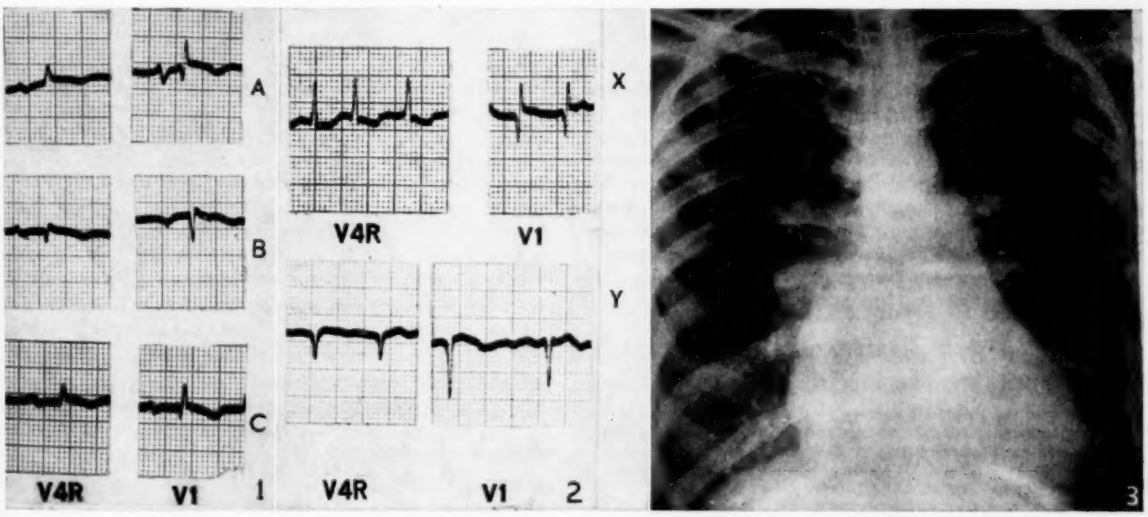

Fig. 1. A=August 1953; B=November 1955; C=August 1957. Electrocardiogram in Case 4, showing right ventricular hypertrophy present in August 1953, absent in November 1955 after successful valvotomy, and again present in August 1957 with the development of re-stenosis.

Fig. 2. X=pre-operative; Y=post-operative. Electrocardiogram in Case 6 showing right ventricular hypertrophy pre-operatively, but no right ventricular hypertrophy post-operatively.

Fig. 3. Teleradiogram of chest in Case 6, showing enlargement of the heart, with prominence of the right ventricle, both atria and the pulmonary artery. The lungs show features of pulmonary hypertension, and 'septal' lines.

noea and paroxysmal nocturnal dyspnoea. Clinically there was evidence of combined mitral stenosis and incompetence. At operation in February 1955, a mobile valve was found with a transverse diameter of 6/8ths inch and a regurgitant stream along the whole length of the valve orifice. The valve was split to 1¼ inch diameter and the surgeon noted no increase of regurgitation. Her effort tolerance has not altered at all since operation, though clinically, the mid-diastolic murmur previously present has disappeared.

Other Causes of Poor Results Despite Adequate Post-Operative Size of the Mitral Valve

In some subjects, adequate surgery is performed but the patients experience little or no improvement. Possible explanations for these cases are:

1. Associated valvular defects, which pre-operatively were considered insignificant or were not diagnosed (e.g. tricuspid stenosis), but were in fact significant.

2. The presence or development of essential hypertension or ischaemic heart disease.

3. Unrecognized rheumatic activity.

4. Persistence of chronic bronchial infections in patients in whom bronchial disease has contributed largely to their disability.

5. The development of permanent atrial fibrillation, not present pre-operatively. We concur with other workers, that atrial fibrillation developing immediately after the operation should be terminated, if possible, by quinidine. In those cases where this is not possible, we feel that 3 repeated attempts at intervals of 3-4 months should be instituted, but that anticoagulant therapy should be given for 10-14 days before each attempt, to diminish the risk of embolization.

6. Persistent pulmonary hypertension. There is no unanimity of opinion about the frequency of persistent pulmonary hypertension after adequate valvotomy. That such a state may prevail in some subjects is agreed. Do such subjects remain incapacitated in terms of limitation of effort tolerance? In our series, pre-operative cardiac catheterization was performed on few cases and post-operative catheterization on an even smaller number. Evans and Short[3] emphasized the importance of right ventricular hypertrophy on the electrocardiogram as a sign of pulmonary hypertension in mitral stenosis. In our series there were 34 patients with electrocardiographic evidence of right ventricular hypertrophy pre-operatively. Of these, 15 had complete regression and 8 had considerable diminution of electrocardiographic evidence of right ventricular hypertrophy. Of the 11 patients in whom right ventricular hypertrophy did not regress, only 4 had valvular splits which would at present be regarded as good and, of these 4, 3 have been much improved, with increase of effort tolerance, the 4th patient having a fair result. The remaining 7 patients had poor splits (4 cases), fair splits (2 cases) and grossly induced mitral incompetence (1 case). In these 7 subjects, 6 had poor results and one, with a fair split, had a good result. It would appear, therefore, that persistent pulmonary hypertension may not be common after a good split and, moreover, that even if present it does not necessarily prevent alleviation of symptoms. Its effect on ultimate prognosis cannot as yet be assessed.

There remains, however, a very small group in whom failure to improve cannot be explained. This group is difficult to investigate without accurate objective methods of assessment of disability.

SUMMARY AND CONCLUSIONS

Mitral valvotomy is an established surgical procedure of low mortality and with a high proportion of good and apparently lasting benefits. However, about 30% of subjects have persistent or recurrent symptoms and the assessment and management of these patients becomes a problem, which is discussed in this article. These patients can be placed in several categories with differing requirements for management. Several of the problems posed by these patients are as yet unanswered. In attempting to assess the reason for continued morbidity, the following questions need to be answered:

1. Is the patient having recurrent bouts of post-commissurotomy syndrome?

2. Was the surgical procedure adequate?

3. Has there been recurrent or continued rheumatic activity?

4. Has re-stenosis occurred?

5. Is there evidence of persistent right heart failure and/or tricuspid incompetence?

6. Was significant regurgitation found at operation?

7. Are associated valve defects, or hypertension, or coronary artery disease responsible for symptoms?

8. Is there persistent chronic bronchial disease?

9. Is atrial fibrillation, not previously present, responsible for disability?

10. Is there persistent pulmonary hypertension?

We wish to express our thanks to Prof. G. A. Elliott, head of the Cardiac Clinic, Johannesburg General Hospital, for permission to undertake this study, and to Mr. A. M. Shevitz for reproduction of the figures. The thoracic surgeons who performed the operations (Mr. L. Fatti, Mr. D. I. Adler, Mr. R. Crawshaw, Mr. D. Fuller and Mr. P. Marchand) are thanked for their cooperation.

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OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL AID SOCIETIES REMOVED FROM THE LIST

The names of the following societies have been removed from the list of approved medical aid societies and the members are no longer entitled to the preferential tariff:

1. Anglo-Alpha (Dudfield) Medical Benefit Society, Private Bag, P.O. Lichtenburg, Transvaal.

2. Hunt, Leuchars & Hepburn Ltd. (Durban) Employees' Medical Benefit Fund, P.O. Box 943, Durban.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
10 September 1958

MEDIESE HULPVERENIGINGS VAN DIE LYS GESKRAP

Die name van die volgende verenigings is van die lys van goedgekeurde mediese hulpverenigings geskrap en lede is nie langer op die voorkeurtarief geregtig nie:

1. Anglo-Alpha (Dudfield) Medical Benefit Society, Privaatsak, Pk. Lichtenburg, Transvaal.

2. Hunt, Leuchars & Hepburn Ltd. (Durban) Employees' Medical Benefit Fund, Posbus 943, Durban.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
10 September 1958

Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

VAN DIE REDAKSIE : EDITORIAL

'N J. S. DU TOIT-GEDENKFONDS

Elders in hierdie uitgawe van ons *Tydskrif* (in die briewe-rubriek) plaas ons 'n brief wat geskryf is deur 'n aantal geneeshere wat almal intieme vriende van wyle dr. J. S. du Toit was. Hulle doen aan die hand dat 'n trustfonds gestig word, wat bekend sal wees as die J. S. du Toit-gedenkfonds, met die vooropgestelde doel om 'n blywende fonds in die lewe te roep waaruit behoeftige seuns en dogters van gestorwe mediese kollegas gehelp sou kon word om hul mediese studies voort te sit.

Die gedagte agter hierdie brief spruit enersyds uit 'n lank gevoelde behoefte aan so 'n fonds; ook spruit dit uit die begeerte om hierdie fonds te koppel aan die naam van dr. du Toit omdat hy so veel van sy kragte geskenk het aan die saak van die medisyne, aan die Mediese Vereniging en aan die beoefenaars van die mediese praktyk self.

Dit is nie hier die plek om dr. du Toit se bydraes tot die medisyne in die algemeen op te som nie. Dit is reeds al gedoen toe van sy vriende sy loopbaan in waarderende herinnering geroep het. Maar, wat tog ter sake is, sou wees om te wys op 'n soortgelyke hulpfonds—die Liefdadigheidsfonds van die Mediese Vereniging, waarvan dr. du Toit die vader en skepper was.

Die Liefdadigheidsfonds van die Vereniging is in die vroeë dae van die Vereniging in die lewe geroep op aandrang van dr. du Toit, met die doel om 'n trustfonds daar te stel waaruit behoeftige afhanklikes van gestorwe dokters gehelp sou kon word. Die fonds is opgebou uit oorspronklike donasies, uit bydraes wat lede van die Vereniging gestuur het en nog stuur aan die fonds, in plaas van om kranse te koop, en uit bemakings. Hierdie fonds is vandag oor die £40,000 sterk en talle weduwees en ander afhanklikes van dokters wat oorlede is, kry bystand uit die fonds. Omdat hierdie fonds 'n spesifieke doel en omvang het, sou hulp vir mediese studies nie inpas by die funksie daarvan nie.

Om hierdie rede het die gevoel nou ontstaan om 'n spesiale opvoedingsfonds in die lewe te roep sodat mediese studente wat hul ouers verloor het, in staat gestel sal kan word om voort te gaan met hul mediese studies—soos hulle sou kon gedoen het indien hul ouers hulle nie ontval het nie.

Afgesien van die groot behoefte aan so 'n fonds, kan ons skaars dink aan 'n waardiger huldeblyk aan 'n groot weldoener van die Mediese Vereniging en van die bewoners van hierdie land, as om ter gedagtenis aan hom 'n opvoedingsfonds van hierdie aard te stig as gedenkfonds en as blywende monument aan sy nagedagtenis.

BIGOTRY IN MEDICINE

Training in medicine and the pressure of work in medical practice after graduation, takes up many years in a man's lifetime and imposes such a discipline on the doctor that it is only one with an unusual character who is able to abstract himself from his traditional way of thought, and who can look upon medicine from the outside.

The genealogical tree of European medicine is a long and honourable one. It follows very closely, although a little behind, general scientific progress. Our medicine was originally Greek; traditionally given from the gods, it was received by Aesculapius, himself half divine, who established famous cults at Cos and Cnidos, and it was Hippocrates, the great teacher of Cos, who codified this knowledge and founded Greek medical science. It has always been believed that Hippocrates was considerably influenced by the teaching of the medicine of Egypt and, as there was a fairly brisk trade between Greece and Egypt, this is undoubtedly possible. If we are looking for true ancestors, the ancient Egyptian physician-priests, deriving as they did many of their teachings from the Sumarians, may be considered the true fathers of our medical way of thought.

From Greece the genealogical tree is clear. Finally codified by Galen, the science passed to the Romans who, more occupied with military than with medical matters, side-tracked much medical development by their extraordinary predilection for inspecting the viscera of animals. Inspection of the viscera of a sacrifice was an ancient custom which they had learned, among many other things, from the Etruscans.

After Galen none in Christendom dared comment critically on medical matters for nearly a thousand years, but Greek medicine was kept alive during the dark ages by the Arabian schools who, when the thousand years were past were to return to the Europe of the Renaissance some of the information they had cherished. From that time on the story of medicine is well known and, while we may well take pride in our ancient lineage, medical thought today seems to be so hidebound that deviation from the accepted way of thinking is considered almost as great a crime as deviation from religious thought was in Reformation times, and as political deviation is in the twentieth century. There must be a reason for this curious medical bigotry.

Various reasons may be put forward to account for this curious involution of our critical faculty. Very few doctors have time to think deeply over the basic principles of medical knowledge, and the personality of our teachers is vested with such extreme authority that their opinions are accepted unconditionally by all of us when students, and the habit persists. Only rarely do we find somebody who has the opportunity of contemplation, and the ability to study what relation medicine, as we practice it, bears to other possible ways of healing.

Western medicine is being practised among barely a quarter

of mankind. China and India contain almost three-quarters of the human race, and they have provided their own medical education and developed their own medical systems from time immemorial. It would be impertinent for an ordinary Western doctor, ignorant as he usually is of what these countries have to offer in the way of medicine, to say that it has no value.

We do know that in India the Ayurvedic system of medicine produces cures which we in the West are unable to explain on a scientific basis; and the study of man and his mind has for many years been a preoccupation of Chinese physicians. On rare occasions a drug from their ancient pharmacopoeia becomes available to us whose action is explainable and whose benefits we appreciate. This is sometimes hailed as a new discovery. Recent examples are the derivatives of *Rauwolfia serpentina*, which has been used for many hundreds of years in India for the control of excitable states and hypertension in the form of extracts of the crude root. In the last decade various alkaloids isolated from it have become available to Western medicine. This is only the latest of a notable line. Quinine furnishes another example. The treatment of malaria by extracts of cinchona bark was known for many years to the people of South America, and the story of its cultivation, extraction and introduction to Europe by the Jesuit missionaries earned it the name of Jesuits' bark.

From the ranks of medical men occasional thinkers do emerge who have the intelligence, courage and energy to flaunt the inhibiting conventions and strike out in a new direction. These are the undaunted pioneers who press towards new developments and are not afraid to meet the challenge of inquiry and research. It is thus of the utmost importance that the aim of medical training should never become rigid and narrow, but should always be directed to developing independent thought.

'THE HISTORY OF MEDICINE IN SOUTH AFRICA'

The first consignment of the *History of Medicine in South Africa*, written by Dr. Edmund Burrows, has arrived in the country. Members will recall that the Medical Association undertook the production of this book as a service to its members and to the country.

All members of the Medical Association of South Africa are entitled to one copy of the work in special de luxe binding at the preferential price of 25s. (The book, bound in cloth, will be available to the general public at 63s.). With the approval of the Medical Association (and in conformity with the practice of learned societies who publish works of importance to give their members an opportunity of receiving preference in obtaining copies), the publisher will send one copy to every member unless the offer has already been turned down. All who do not wish to keep it will have the opportunity to return it after inspection and within a limited time; we hope that this will not be necessary.

This book is the first comprehensive account of the historical development of medical practice in South Africa and traces the fascinating story of the struggle against disease and ill-health in this country from its beginnings to the end of the nineteenth century.

Sponsored by the Medical Association of South Africa, this work can be regarded as the official history of medicine in South Africa. The Association confidently expects that every member will wish to retain such a valuable piece of Africana.

CHLOROTHIAZIDE—ITS MODE OF ACTION AND ITS USE IN PATIENTS WITH OEDEMA¶

S. J. SAUNDERS, M.B., CH.B., F.C.P. (S.A.) and L. EALES, M.D., M.R.C.P.

From the Laboratory of the Department of Medicine, University of Cape Town and Groote Schuur Hospital, Cape Town

The development of an effective oral diuretic is an important step forwards in therapeutics. To date the most effective diuretic agents have been the organic mercurial compounds administered by intramuscular injection. These have been associated with remarkably few side-effects. The very small mortality rate has occurred generally with intravenous administration.[1-4] Undesirable effects of the mercurial diuretics are the development of the salt-depletion syndrome and of hypokalaemic alkalosis, but the major disadvantage is the necessity of intramuscular injections. These compounds when given by mouth are less effective diuretics while increase in the dosage results in toxic effects, particularly in respect of the gastro-intestinal system. Axelrod and Pitts[5] have suggested that the mercurial diuretics specifically block the renal tubular reabsorption of chloride. There is an associated loss of sodium and water but not of bicarbonate. The potassium loss is variable.

A completely different biological approach was used with the introduction of acetazoleamide,* a heterocyclic unsubstituted sulphonamide. It inhibits carbonic-anhydrase activity. This enzyme normally catalyses the conversion of carbon dioxide and hydrogen to carbonic acid. When this action is inhibited in the renal tubule there is depression of sodium and hydrogen-ion exchange resulting in an increased excretion of bicarbonate, while titratable acidity and ammonia diminish or disappear. In addition, potassium normally competes with hydrogen for excretion and during acetazoleamide administration the reduction in hydrogen-ion secretion is associated with an increased tubular secretion of potassium.[6, 7] Acetazoleamide administration therefore results in the excretion of an increased volume of alkaline urine rich in bicarbonate without an increase in chloride. *Pari passu* a state of hyperchloraemic acidosis develops. Since there is a reduction in the amount of bicarbonate filtered by the glomeruli,[8] with continued administration acetazoleamide soon loses its effect in so far as bicarbonate excretion is concerned. The reduced capacity to exchange hydrogen for sodium, however, persists, thus preventing the correction of the acidosis. In our own experi-

* Diamox.

ence and that of others[9, 10] it has been an unsatisfactory diuretic.

Yet another series of diuretic agents in current use is the amino-uracils. Aminometradine† is an oral diuretic which is considered to act by directly interfering with the tubular reabsorption of sodium and/or chloride. Anorexia, nausea and vomiting are frequent effects.[11] More recently amisometradine‡ has been on trial.[12] It appears to be less liable to produce gastro-intestinal effects, but in our limited experience has not been as effective as the organic mercurials.

Chlorothiazide¶ is 6-chloro-7-sulphamyl-1, 2, 4-benzothiadiazine-1, 1-dioxide—a substituted benzothiadiazine compound with a free sulphonamide group (Fig. 1). It was first synthesized by Novello and Sprague.[13] It was found to be

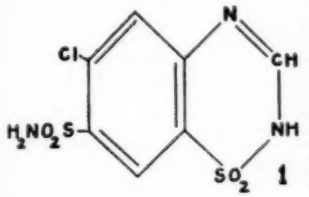

Fig. 1. Chlorothiazide (6-chloro-7-sulphamyl-1, 2, 4-benzothiodiazine-1, 1-dioxide). The detailed structure of the heterocyclic ring of chlorothiazide is not known with certainty, although available evidence favours a structure with the 3, 4 double bond as the predominant form.

a potent carbonic-anhydrase inhibitor *in vitro*, yet the drug in animals has produced a large and almost equimolecular excretion of sodium and chloride without producing acidosis or alkalosis.[14]

In a preliminary report[15] we confirmed that the mode of action of chlorothiazide was similar to that of the organic mercurial compounds inasmuch as its major action was a tubular one increasing the urinary excretion of chloride. Despite its known *in vitro* action as an inhibitor of carbonic anhydrase, the slight increase in urinary excretion of bicarbonate indicated but slight activity in this respect in man. Several favourable reports on its use as an oral diuretic have appeared.[16-21]

This communication is concerned with (i) detailed short-term studies on the mode of action and (ii) the results of clinical experience in 33 patients with oedema.

I. SHORT TERM STUDIES ON MODE OF ACTION

Methods

Studies were carried out in 3 patients. They included one with hypertensive congestive heart failure (No. 18, Table II) and 2 cases of the nephrotic syndrome (Nos. 22, 23, Table IV). All 3 patients had long-standing oedema.

After adequate control periods 2 g. of chlorothiazide were given by mouth. The effect on the glomerular filtration rate and the renal excretion of the electrolytes was followed for 2½ hours in one patient, for 4½ hours in another, and for 7 hours in the third. The duration of the periods was approximately 20, 30 and 60 minutes respectively. Urine was collected by means of indwelling catheters. The bladder was washed out with a known volume of sterile distilled water and air insufflation ensured complete emptying of the bladder. The urine was collected under toluol and then frozen. Blood was drawn 2½ minutes before the mid-point of each period. Serum under oil was centrifuged and separated immediately and then analysed promptly or stored frozen. In the first 2 patients the glomerular filtration rate was measured as the clearance of insulin, constant serum levels being maintained by means of a constant infusion apparatus. In the third patient endogenous creatinine clearance was used as a measure of the glomerular filtration rate.

Biochemical Methods. For sodium and potassium, flame photometry with a Barclay flame photometer using lithium as an internal standard. For chloride, Schales and Schales' method.[22] For carbon dioxide, urinary ammonia, and titratable acidity, Peters and Van Slyke's method.[23] For insulin, Schreiner's method.[24] For creatinine, Bonsnes and Taussky's method.[25] Blood urea and serum proteins were determined by standard laboratory methods of the Department of Chemical Pathology, University of Cape Town.

Results

In all 3 cases the administration of chlorothiazide produced marked alterations in the electrolyte pattern of the urine. The changes in cases 18 and 23 are illustrated graphically in Figs. 2 and 3. The most striking alterations are in the urinary excretion of sodium and chloride, the chloride excretion exceeding the sodium. Fig. 4 illustrates the events in case 18. The increase in urinary chloride begins within ½ hour and there is a stepwise increase until a peak is reached 2½ hours after the administration of chlorothiazide. Thereafter the excretion continues at a high though gradually decreasing level throughout the duration of the experiment. Sodium behaves similarly, but potassium increases to a lesser extent (Fig. 5). Urine volume shows a similar increase. Further observations have shown that this enhanced excretion continues for a variable period of time, which is commonly up to 8 hours. In case 18, the glomerular filtration rate remained unchanged (Fig. 4), while in case 23 there was a slight rise in the glomerular filtration rate during the first and second periods following chlorothiazide. Case 22 was followed for 7 hours with hourly periods, and similar electrolyte changes

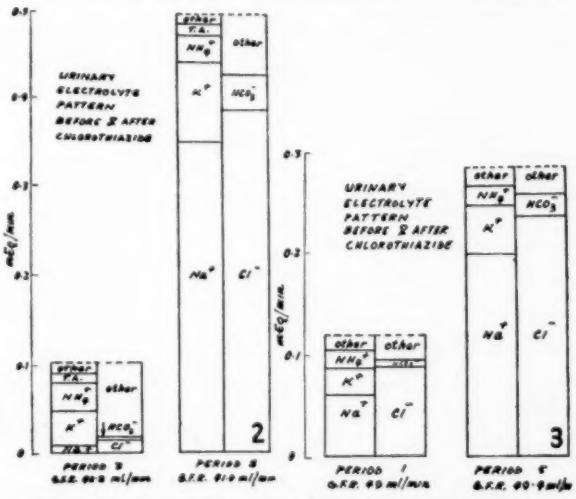

Fig. 2. Case 18. This illustrates the marked changes in urinary electrolyte excretion after 2 g. of chlorothiazide in a patient with hypertensive heart failure. The glomerular filtration rate is comparable in the two periods.

Fig. 3. Case 23. The changes in urinary electrolyte excretion are less pronounced in this case of the nephrotic syndrome. The glomerular filtration rate is comparable in the two periods.

† Mictine. ‡ Rolicton. ¶ Chlotride, Saluric and Diuril.

were encountered without significant alteration in the glomerular filtration rate.

Fig. 6 illustrates the changes in case 18 in the bicarbonate titratable acidity and in urinary ammonia following the administration of chlorothiazide. There was a rise in the urinary pH by the second period in case 18 and a temporary rise during the first period in case 23

Thus of the cations, apart from sodium, there was a lesser increase in potassium while there was little change in ammonia and a slight fall in titratable acidity. Of the anions the striking chloride increase completely overshadowed the slight rise in urinary bicarbonate. There was no increase in phosphate excretion in case 18.

Conclusion. These figures illustrate that the mode of action of chlorothiazide is primarily on the renal tubule, resulting in an increase in the excretion of chloride, sodium, and water. The excretion of sodium and chloride is almost equimolecular although chloride usually exceeds sodium. The potassium excretion increases to a lesser extent. There is a slight increase in the urinary bicarbonate and an associated rise in the pH of the urine.

II. CLINICAL EXPERIENCE IN 33 PATIENTS WITH OEDEMA

All patients, except 2, were admitted to the wards of Groote Schuur Hospital. Judgment of the effectiveness of chlorothiazide was made by clinical assessment, by observation of change in weight, and by urine output in relationship to fluid intake. In some cases sodium, potassium and chloride excretion was measured. A careful watch was kept on the familiar pitfalls of routine ward collections of 24-hour urine samples. In assessing the action of a diuretic, the effects of bed rest, diet and previous therapy had to be considered; for example, considerable diuresis and loss of weight while at rest was noted in one patient before the administration of chlorothiazide. The diet was salt-restricted (±30 mEq. per 24 hours) except in case 4, where the salt intake was unrestricted.

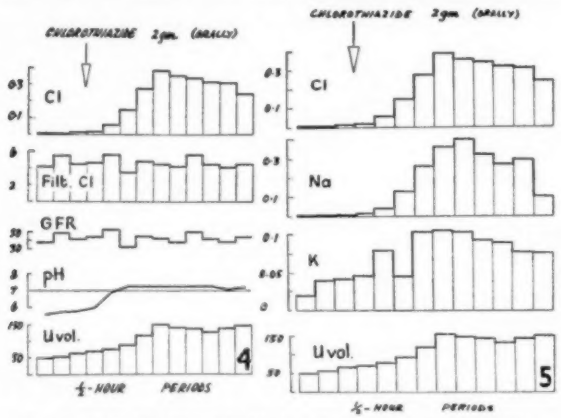

Fig. 4. Case 18. Cl=chloride in urine, mEq./min. Filt. Cl=Filtered load, mEq./min. GFR=glomerular filtration rate (ClN), ml./min. U vol.=urine volume, ml. The chloride excretion increases in stepwise fashion after the administration of 2 g. of chlorothiazide. The glomerular filtration rate and the filtered load of chlorine remain relatively constant. The rise in pH is illustrated on this chart. (The true urine volume is less 40 ml./period—the volume of bladder rinse.)

Fig. 5. Case 18. Cl=chloride in urine, mEq./min. Na=sodium in urine, mEq./min. K=potassium in urine, mEq./min. U vol.=urine volume, ml. The sodium and chloride excretions follow each other. There is a slight increase in potassium excretion as well. The potassium scale is larger than that of the sodium and the chloride.

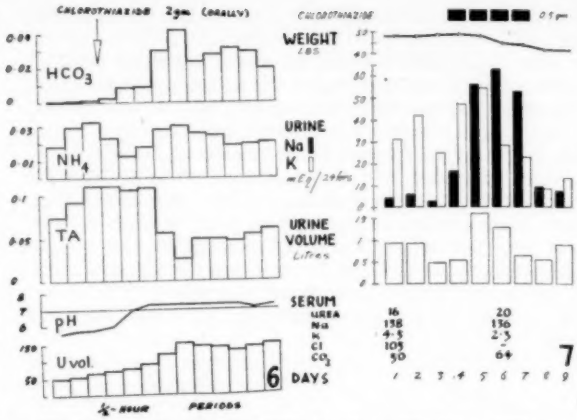

Fig. 6. Case 18. HCO_3=HCO_3 in urine, mEq./min. NH_4=ammonia and amino acids in urine, mEq./min. TA=titratable acidity, mEq./min. U vol.=urine volume, ml. This illustrates the slight changes in urinary bicarbonate, ammonia and titratable acidity following chlorothiazide.

Fig. 7. Case 28, nephrotic syndrome: Prompt increase in urinary sodium and potassium followed 0·5 g. of chlorothiazide. On the first day potassium loss exceeded sodium loss. Note the rapid development of hypokalaemia.

The daily dosage used is indicated in Tables II, III and IV. Varying dosage schemes were used. The results in the 33 patients are summarized in Table I. The relevant clinical features and the response in the patients with cardiac, hepatic and renal oedema are summarized in Tables II, III and IV. Examples of effective chlorothiazide therapy are illustrated in Figs. 7, 8, 9 and 10.

TABLE I. RESULTS OF TREATMENT

Aetiology	*No. of Patients*	*Good Response*	*Fair Response*	*Doubtful Response*	*No Response*
Cardiac Oedema (Table II)	21	13	2	2	4
Renal Oedema (Table III)	10	4	2	—	4
Hepatic Oedema * (Table IV)	2	2	—	—	—
Total	33	19	4	2	8

* One with associated constrictive pericarditis

Of the 21 cases with cardiac oedema (Table II) 11 were due to rheumatic heart disease, 3 to each of ischaemic heart disease and hypertensive heart disease, and 1 to each of syphilitic aortic incompetence, subacute cor pulmonale, chronic cor pulmonale, and right heart failure in a grossly obese subject with pulmonary hypoventilation. There was a good response in 13 patients; this was assessed by significant weight loss, by a diuresis of up to 3 litres in the succeeding 24 hours, sodium loss of up to 400 mEq. over the same time, and clinical improvement. In 2 the response was assessed as fair, in 2 doubtful. In 4 patients there was no response at all; 3 of these had chronic rheumatic valvular disease with gross congestive heart failure (8, 9 and 11) and the 4th (16) had intractable congestive failure following myocardial infarction. Cases 9 and 11 were moribund at the time of treatment. Case 8, who was in acute congestive heart failure,

TABLE II. CARDIAC OEDEMA

No.	Race	Sex	Age	Diagnosis	Oedema	Duration of oedema	JVP cm. above angle of Louis	Creps. at bases	Liver enlargement, finger-breadths down	Serum albumin g./100 ml.	Blood urea mg./100 ml.	Dose, g. 24/hr	Maximum sodium loss mEq./24 hr	Loss or gain in weight lb./days	Response	Remarks
1	E	F	42	Rh., MI	++	2 yr	10+	+	3	3·8	51-23	1-2 (*b*)	160	−7¼/12	Good	Previously refractory to Mersalyl.
2	C	M	44	Rh., MS	++	mths	5	+	2	3·7	40-?	2x	—	−12/10	Good	Poor response to Mersalyl before adm.
3	E	M	58	Rh, MI, AI	+	5 mth (*a*)	10+	+	3	3·8	43-35	1	—	−7¼/8	Good	Refractory to Mersalyl before adm.
4	E	F	47	Rh, MI, AI	+	mths	6	++	2	4·3	33-12	2 (*c*)	166·6	−7/8	Good	Complicating S.B.E. suspected.
5	C	M	48	Rh, MS, AS, AI	++++	1 wk	15	+	1	3·1	67-60	1	—	−8/4	Good	Complicating S.B.E. suspected.
6	C	M	54	Rh, MS, MI	++++	8 mth	10+	—	4	2·8	16-32	2 (*b*)	433	−11/7	Good	See Fig. 8.
7	E	M	52	Rh, MI, AI	++++	mths	10+	++	5	3·4	?-130	2-1	—	−4½/3	Good	Later refractory to 5 g. daily.
8	C	M	13	Rh, MS, MI	++++	2 wk (*a*)	10+	++	5	—	18-50	1-2 (*b*)	56	0/4	None	Responded better after Mersalyl.
9	E	F	40	Rh, AS	++	1 mth + (*a*)	4	++	2	3·6	35-91	2 (*c*)	—	+2/10	None	Refractory to all treatment.
10	E	M	44	Rh, AI	++++	yrs	5	++	4	2·2	102-158	2 (*d*)	—	−2½/7	Fair	Refractory to all treatment.
11	C	F	67	Rh, MI	++	2 mth (*a*)	10+	++	2	3·8	81-32	2 (*c*)	—	+1¼/9	None	Previously poor response to Mersalyl.
12	C	M	51	S. AI	+	2 wk	5	+	3	—	103-39	2 (*c*)	—	−2½/11	Fair	
13	E	M	49	Chron. CP	++++	3 yr	10+	++	4	—	61-37	1	—	−7/6	Good	Previously poor response to Mersalyl.
14	E	F	53	PE, CP	+	1 wk	4	++	3	3·8	10-20	1	—	−2½/7	Good	Possible underlying fibro-elastosis.
15	E	M	54	M Inf.	++	1 yr	4	—	1	4·4	55-52	1	—	−6/14	Good	Claimed better than Mersalyl.
16	E	M	60	M Inf.	++	mths	10+	++	3	4·4	38-?	1	—	+2/7	None	Refractory to Mersalyl.
17	E	M	54	IHD	+	2 wk	6	++	2	3·9	58-51	1 (*d*)	—	−5½/5	Good	Poor response to Mersalyl.
18	E	M	51	EH	++++	mths	10	++	3	3·7	30-50	2 (*d*) (*b*)	—	−10½/6	Good	No effect on hypertension.
19	C	M	12	MH, URD	++++	mths	10+	++	3	—	28-?	2	—	−1/2	Doubtful	Later nephrectomy performed.
20	C	F	46	MH	++	4 mth	4	++	3	3·8	33-34	1	—	−1/2	Doubtful	Diuresis 3 litres after single dose.
21	E	M	51	HS, O	++	8 yr	6	++	?	3·9	41-51	2 (*d*)	—	−11/8	Good	KCl therapy because long administration.

TABLE III. HEPATIC OEDEMA

No.	Race	Sex	Age	Diagnosis	Oedema	Duration of oedema	JVP cm. above angle of Louis	Creps. at bases	Liver enlargement, finger-breadths down	Serum albumin g./100 ml.	Blood urea mg./100 ml.	Dose, g./24 hr	Maximum sodium loss mEq./24 hr	Loss or gain in weight lb./days	Response	Remarks
32	C	M	30	Cirrhosis of the Liver	++++	3/12	9	+	1	2·7	26	2 (*d*)	160	−4½/7	Good	Liver biopsy confirms diagnosis.
33	C	M	21	Parenchymatous Liver Disease with Constrictive Pericarditis	++++	3/12	12+	++	3	2·0	12	2-1-2 (*d*)	122·5	−7/6	Good	

TABLE IV. RENAL OEDEMA

No.	Race	Sex	Age	Suspected Aetiology	Oedema	Duration of oedema	Hypertension	Serum albumin g./100 ml.	Serum cholesterol mg./100 ml.	Haematuria microscopic	Blood urea mg./100 ml.	Dose, g./24 hr	Maximum sodium loss mEq./24 hr	Loss or gain in weight lb./days	Response	Remarks
22	B	F	29	Type II	++++	1 yr	No	2·5	682	No	78-43	2 (*d*)	116	−11¾/14	Initially good	Later refractory to all therapy.
23	E	F	41	Type II	++++	4 mth	No	2·3	480	No	48-50	2	93·1	−5/7	Initially good	Later refractory to all therapy. Fall in serum potassium from 4·2 to 3·2.
24	E	F	12	Type II	++	5 mth	No	2·7	357	No	21-?	1-2 (*d*)	—	+¾/12	None	Refractory to steroid therapy.
25	C	F	53	Type II	++++	1 mth	No	2·7	605	No	40-?	2 (*b*)	—	+1½/7	None	Refractory to all therapy.
26	E	M	2	Type II	++++	3 wk	No	0·6	760	No	25-42	½ (*d*)	—	+1½/6	None	Refractory to all therapy.
27	E	F	2	Type II	++++	4 mth	No	1·9	550	No	17-53	1 (*d*)	—	−¾/5	None	Refractory to all therapy
28	E	F	6	Type II	++	+1 yr	Yes	0·9	840	Yes	20-40	½	63·1	−6½/6	Good	Hypokalaemic alkalosis developed.
29	C	M	32	Type I	++	10 mth	Yes	2·3	370	No	47-94	1 (*e*)	—	−3½/7	Good	Confirmed by renal biopsy.
30	C	M	29	Type I	++++	1 mth	No	2·8	395	Yes	253-255	1	—	−2½/5	Fair	Autopsy confirms diagnosis.
31	E	M	53	Dissd. lupus eryth.	++++	9 mth	Yes	2·3	567	Yes	50-64	2	52·5	−1¼/4	None	Died 3 months later with massive oedema. Refractory to all treatment.

(*a*) = Previously in CCF. (*b*) = Intermittent omission of daily dose. (*c*) = Every third day (*d*) = In 2 doses, a.m. and p.m. (*e*) = Alternate days.

JVP = Jugular venous pressure. Rh = Rheumatic heart disease. MI = Mitral incompetence. MS = Mitral stenosis. AI = Aortic incompetence. AS = Aortic stenosis. S = Syphilis. CP = Cor pulmonale. PE = Pulmonary embolism. M Inf = Myocardial infarction. IH = Ischaemic heart disease. EH = Essential hypertension. MH = Malignant hypertension. UR = Unilateral renal disease. HS = Hypoventilation syndrome. O = Obesity, Types I and II = Types I and II nephritis. SBE = Subacute bacterial endocarditis. Under Race, E = European, C = Coloured, B = Bantu.

Where 2 blood-urea figures are given they refer to values before and after treatment. —

showed no weight alteration, an unimpressive diuresis, but a sodium loss of 58 mEq. in 24 hours.

Of 10 cases with the nephrotic syndrome (Table IV) 7 were due to type-II nephritis, 2 to type-I nephritis, and 1 to disseminated lupus erythematosus. The response was good in 4 and fair in 2. In 4 there was no response at all; there was no obvious reason for this; the serum-electrolyte concentrations were normal but measurements of the glomerular filtration rate were not available in most of these patients.

One patient with cirrhosis of the liver responded well, and another with constrictive pericarditis and cirrhosis of the liver also responded well (Table III).

Two cases with the nephrotic syndrome (22 and 23) and 2 with cardiac oedema (7 and 15) responded initially and later became refractory. Four patients with cardiac oedema (1, 2, 3 and 13) were clearly resistant to mersalyl and responded well to chlorothiazide. Case 25 with the nephrotic syndrome had responded to mersalyl but did not respond to chlorothiazide. Case 8 with cardiac oedema responded moderately well to mersalyl but had had chlorothiazide on the previous day—during which he excreted 56 mEq. of sodium. A potentiating action is a possible explanation. This also might have been the case in one other patient.

There were no side-effects. No patients complained of anorexia, nausea or vomiting and there was no evidence of leucopenia or liver or renal damage except for a rise in blood-urea concentration in some of our patients. Two of these patients, with congestive heart failure, were moribund and the 3rd had disseminated lupus erythematosus. In 3 patients with the nephrotic syndrome daily measurement of the protein loss in the urine showed no alteration during chlorothiazide therapy.

Studies of serum electrolytes in most of the patients before and after treatment showed little change. A fall in serumpotassium concentration was noted in 9 patients and was moderate except in cases 28 and 32, where levels of 2·3 and 2·6 mEq. per litre were recorded at the end of therapy.

(Apart from the use of chlorothiazide in patients with oedema we have administered the drug to one patient with periodic paralysis and a few patients with hypertension.

In the case of periodic paralysis the administration of 1 g. of chlorothiazide after 42 days on a low-sodium diet (26 mEq. per day) resulted in the loss of 270 mEq. of sodium in the urine. The administration of 0·5 g. of chlorothiazide to 3 normal subjects on a similarly sodium-restricted diet resulted in a loss of sodium, potassium and chloride in the urine.[26] The sodium loss in the 3 subjects amounted to 64, 155 and 69 mEq. respectively.

Our experience with the use of chlorothiazide in the management of hypertension is too limited to state any definite conclusions. In 6 patients with essential hypertension the drug alone appeared to be associated with a significant fall in blood pressure in only one and, in that instance bed rest and a low-sodium diet had been maintained for only 1 week. In 2 other patients receiving mecamylamine the administration of chlorothiazide was associated with a further fall in blood pressure and the dose of mecamylamine had to be reduced. Harrington *et al.*[27] have ascribed this effect to possible diminution in excretion of mecamylamine.)

DISCUSSION

Beyer *et al.*[14] have stated that *in vitro* studies have shown that chlorothiazide is 30 times more potent a carbonic-anhydrase inhibitor than acetazoleamide and therefore it is surprising to find that in man its action in this respect is extremely poor. It resembles the mercurial group of drugs in that its prime effect is on the renal tubule with excretion of chloride and sodium. Others have confirmed these effects. In addition we have noted the rapid development of hypokalaemic alkalosis following chlorothiazide therapy, an effect which again resembles the action of the mercurial diuretics.

There are, however, points of difference. We have noted possible potentiation of the action of the mercurial diuretics, and, more important, in some cases chlorothiazide was effective when mersalyl had failed. While the effects on the

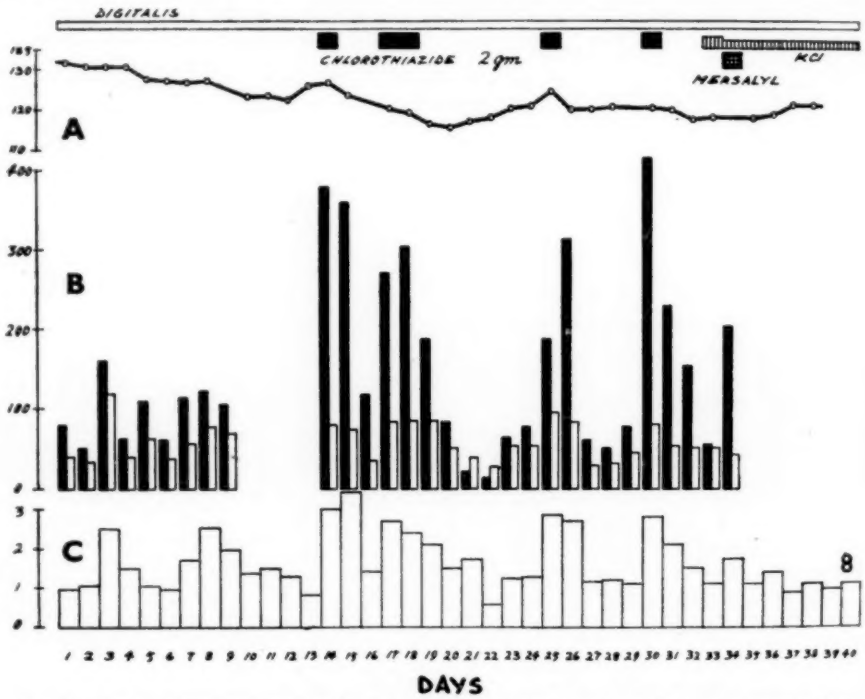

Fig. 8. Case 6. Congestive cardiac failure—mitral valve disease. A=weight, lb. B=urine Na (black columns) and K (white columns), mEq./24 hrs. C=urine volume, litres. Digitalis therapy was associated with a loss of sodium, but the addition of chlorothiazide in 2·0 g. dose was associated with a striking increase in urinary sodium to as much as 400 mEq. per day. The sodium loss from single doses appeared to continue for at least 48 hours. Potassium excretion was relatively unaffected although serum potassium had fallen to 3·6 mEq. per litre.

excretion of water, sodium, chloride and potassium are very similar to those which are encountered after the administration of the organic mercurial diuretics the rise in urinary pH is unlike mercurial action. In addition Beyer[28] states that chlorothiazide is markedly active under experimental conditions of sodium bicarbonate alkalosis, while the mercurial diuretics are essentially ineffective under these conditions. Furthermore 'BAL'* has no effect on chlorothiazide diuresis whereas it markedly depresses the sodium and chloride excretion induced by the mercurial diuretics. Laragh *et al.*[17] has noted a further point of difference in that 'free' water clearance is increased in mercurial-induced diuresis but not in chlorothiazide-induced diuresis. Apart from these differences we have observed that in case 22, where amisometradine failed to induce a diuresis when given in adequate dosage (up to 3·2 g. daily), chlorothiazide was immediately effective. It is possible that its mechanism of action on the renal tubule differs not only from that of the mercurial diuretics but also from that of the amino-uracil diuretics.

The diuresis starts within 2 hours of administration of the drug and the duration of action usually extends maximally over the next 8 hours, but there seems to be a prolongation of the effect for a full 24 hours and in an occasional case to 48 hours (Fig. 8). While some have recommended that the dose should be divided into a morning and an afternoon dose, this method of administration does not appear to us to have any advantage over a single dose administered in the morning. A single dose of 1·0-2·0 g. was administered daily in 12 adult patients. In 8 of them 1·0 g. daily produced an adequate response, while in 4 others 2·0 g. daily appeared to be more satisfactory. However, 1·0-2·0 g. every third day appeared to be an effective therapeutic regime in certain patients and carried less risk of hypokalaemia. The unsupervised prolonged use of this drug may be dangerous; and dosage may well have to be altered repeatedly to suit individual patients.

It is obvious that chlorothiazide is an oral diuretic which is effective in many cases of cardiac, renal and hepatic oedema. In our judgment it is frequently as effective as the organic mercurials administered parenterally in their usual doses. In this series of 33 patients the drug appeared to be devoid of toxic effects. The rapid development of hypokalaemic alkalosis, especially when the drug is given daily, should be borne in mind (Fig. 7). While Laragh *et al.*[17] noted that mild to moderate hypokalaemic alkalosis developed in most of their 32 patients, this complication was less frequently encountered in our patients. This may be attributed to the fact that, whereas their patients were maintained on a diet rigidly restricted to not more than 12 mEq. of sodium per 24 hours, our patients were receiving not more than 30 mEq. per 24 hours. Sherlock *et al.*,[29] however, have reported the occurrence of hypokalaemia in patients with cirrhosis of the liver and ascites on a dietary sodium intake of 22 mEq. daily, while Bayliss *et al.*[30] state that hypokalaemia might develop during intermittent treatment when chlorothiazide is given on 4 consecutive days each week, and could be induced in patients having a sodium intake of 214 mEq. per day! On occasions, however, in some of our patients, potassium loss exceeded the sodium loss (Fig. 10). In cases with marked sodium depletion it is to be expected that the predominant cation excreted would be potassium rather than sodium. Hypokalaemia may be dangerous, not only in itself, but also by increasing sensitivity to digitalis. Hypokalaemia can be corrected by potassium supplementation which, however, should be administered with caution to markedly oedematous patients with hyponatraemia. The wider spacing of doses, viz., 1-2 g. every third day or 1-2 g. daily for, say, 3 days followed by a similar period without treat-

* British Antilewisite.

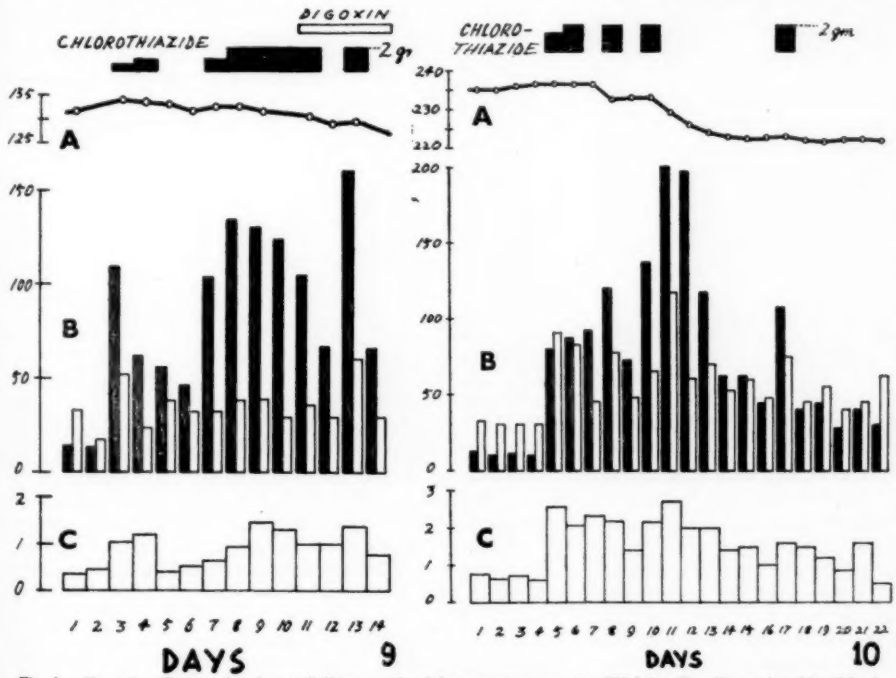

Fig. 9. Case 1. Congestive heart failure—mitral incompetence. A=Weight, lb. B=urine Na (black columns) and K (white columns), mEq./24 hrs. C=urine volume, litres. Even with the initial dose of 0·5 g. of chlorothiazide the marked and prompt increase in the urinary sodium loss is evident. 2·0 g. produced a pronounced naturesis. The potassium excretion increased only slightly.

Fig. 10. Case 22. A=Weight, lb. B=urine Na (black columns) and K (white columns), mEq./24 hrs. C=urine volume, litres. The prompt effect of chlorothiazide on sodium and water excretion in a patient with the nephrotic syndrome is clearly evident. Note, too, the marked increase in potassium loss. This patient later became refractory to chlorothiazide.

ment, may be advisable. In cases receiving protracted heavy dosage the development of listlessness, apathy, weakness and diminution or loss of tendon reflexes should be watched for, serum electrolyte concentrations should be determined, and monitoring with the electrocardiogram for the changes of hypokalaemia is advisable.

There appear to be a few contra-indications to the use of chlorothiazide in oedematous patients, as follows: (1) Pre-existing hypokalaemia and (2) marked elevation of the blood-urea concentration (although several of our patients with blood ureas ranging between 50 and 100 mg.% received chlorothiazide without obvious deterioration). (3) In patients with cirrhosis of the liver and ascites it should be administered with caution. Sherlock *et al.*[29] have emphasized the development of hypokalaemia in such patients with, in some, the appearance of hepatic foetor, tremor, confusion and, in one case, the loss of consciousness—the usual features of hepatic pre-coma and coma.

It is clear that certain patients with oedema who responded well initially became refractory to treatment. The reasons for this are not apparent. Certain patients who were refractory to all other diuretic therapy did not respond to chlorothiazide even in doses of as much as 5·0 g. daily. We have little reason to recommend a dose above a maximum of 2·0 g. daily.

SUMMARY

1. Short-term studies in 3 patients suggest that chlorothiazide acts by inhibiting the renal tubular reabsorption of chloride and sodium. In this it resembles the organic mercurial diuretics, but there is a rise in urinary pH and a slight increase in the urinary bicarbonate.

2. Clinical experience in 33 patients with oedema is reported. The results indicate that in a high proportion of cases it is an effective oral diuretic. A maximum daily dose of 1-2 g. is advisable in the adult. This may be given as a single dose or twice daily.

3. In this series of patients chlorothiazide administration was devoid of toxic effects. The main complication is hypokalaemia, and this may necessitate supplementary potassium chloride and the adjustment of the dosage schedule to the requirement of a particular patient. Prolonged unsupervised use of the drug is to be avoided.

4. Patients may become refractory to chlorothiazide after having previously responded well.

One of us (L.E.) is in receipt of assistance through the Clinical Nutrition Research Unit supported in the Department of Medicine, University of Cape Town, by the Council for Scientific and Industrial Research.

Acknowledgment is made to Prof. G. C. Linder for his advice and help; to Misses M. Fischdick, J. Sutherland and J. Seward, B.Sc. for their assistance; to Mr. R. Thompson; to the consultant staff for access to their patients; to Dr. S. Burger, the Medical Superintendent of Groote Schuur Hospital, for facilities and to Messrs. Merck, Sharp and Dohme for generous supplies of chlorothiazide.

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THE EMERGENCY TREATMENT AND TRANSPORT OF PATIENTS WITH RESPIRATORY FAILURE

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Acute respiratory failure may occur in a number of conditions and may vary in severity from a minor degree of impairment of respiratory exchange to complete cessation of breathing. The most obvious and serious concomitant of such failure is anoxia, with all of its secondary manifestations on the cardiovascular and central nervous systems. Less often thought of, but yet of as great importance, is the consequent inadequate elimination of CO_2, which may produce effects as disastrous as those due to lack of oxygen.

The conditions most commonly associated with acute respiratory insufficiency are the following:

1. Poliomyelitis involving the muscles of respiration and/or the muscles of the pharynx and larynx.
2. Polyneuritis, e.g. infective and that due to porphyria.
3. Head injuries.
4. Injuries of the upper cervical region of the spinal cord.
5. Poisoning, e.g. from narcotics, insecticides of the anti-cholinesterase group, shellfish which have ingested poisonous plankton (see below).
6. Respiratory paralysis deliberately induced as a therapeutic measure, e.g. in cases of tetanus.

The occurrence of recent cases[1,2] of fatal paralytic *mussel poisoning* has re-awakened interest in this uncommon condition and has shown that here is another cause of acute respiratory failure which the practitioner may be called upon to treat. Mussels and clams may become poisonous when they have strained the dinoflagellate plankton *Gonyaulax* out of the water; the poison has been shown to be a neuromuscular toxin with effects on both skeletal muscle (curare-like) and peripheral nerves.[3] In addition the poison is said to have a

depressant action on the myocardium and its conducting tissues.[4] As there is no known antidote to this poison the treatment remains purely symptomatic and with this in mind it might be of help to have a clear regime to follow in the cases showing signs of respiratory failure.

It is important from the outset to bear in mind that in patients apparently suffering from respiratory failure there are two equally vital functions to be considered. These are (1) the mechanism responsible for the provision of pulmonary ventilation—i.e. the diaphragm and the intercostal muscles, and (2) the mechanism responsible for the protection of the respiratory tract from foreign material—the pharyngeal and laryngeal mechanisms. Either one or both of these may be affected, according to the nature and extent of the condition responsible, thus giving rise to the three broad categories described by Crampton Smith, Spalding and Russell,[5] each of which requires different management.

I. *Patients with paralysis or weakness of the muscles of ventilation only*

Here there is involvement of neither the larynx nor the pharynx, so that swallowing is intact and the patient can prevent the entry of foreign material into the respiratory tract. Respiratory exchange however, is inadequate, and a varying degree of suboxygenation will be present, depending on the degree of paralysis. Retention of CO_2 is also likely and again its extent depends upon the degree of underventilation. The patient in fact, is suffering from both lack of oxygen and accumulation of carbon dioxide and it is necessary to correct both of these.

If the paralysis is incomplete the administration of an oxygen enriched atmosphere will help to correct the anoxic element, but will do nothing to correct the CO_2 retention; it is important to realize that CO_2 itself, when present to excess, may eventually cause complete respiratory failure through its depressant effect on the respiratory centre.

In cases of more extensive paralysis, even the administration of pure oxygen will not correct the anoxia and it is quite futile to administer oxygen if the patient's lungs are not adequately ventilated with it. Only too often does one see the patient with markedly impaired respiratory ventilation being 'given oxygen', which ineffectively fans the face and does little to correct the oxygen deficiency of the haemoglobin and nothing to correct the CO_2 accumulation which is taxing the pH-regulating mechanism to its utmost. These patients need *active mechanical ventilation* to make up for the inadequacy of their own diaphragms and intercostal muscles and, if such ventilation is adequate, there should be no need even to administer oxygen—air is quite good enough and the CO_2 retention will be corrected. Additional oxygen should only become necessary when pulmonary collapse or pneumonia or interference with diffusion across the alveolar membranes complicates the picture.

II. *Patients with involvement of the protective mechanism only*

Cases of bulbar palsy are frequently treated incorrectly, with disastrous results. The muscles of ventilation are acting adequately, but there is disorganization of the pharyngeal and laryngeal mechanisms, so that saliva or anything else that may be in the pharynx tends to enter the trachea. This may lead to a variety of complications, from atelectasis due to plugs of mucus to severe acute pulmonary oedema due to regurgitated acid gastric juice, but severe pulmonary infection is the most frequent result and often the ultimate cause of death. Because the vocal cords cannot close, an effective cough is not possible and severe respiratory distress rapidly ensues because insufficient alveolar surface is available for normal gas exchange.

Treatment here is aimed firstly at prevention, by early accurate assessment of the condition. Pooling of secretions in the pharynx or trachea is usually accompanied by a characteristic rattling sound on respiration. Movement of the thoracic cage will be good. Phonation may be impaired, the voice being weak or merely a whisper. Attempts to swallow show that movement of the palate and pharyngeal muscles is weak or absent and dribbling of saliva may be evident.

At this stage, if little or no foreign material has entered the respiratory tract, treatment consists of providing protection only. This is adequately secured by posture alone, it being best to place the patient in a head-down position, lying on his side or prone but never on his back, for this will cause pooling of secretions in the posterior pharynx with eventual spill into the trachea. No active ventilatory assistance need be given, as the diaphragm and intercostals will be working well. If, on the other hand, the patient has already inhaled a fair amount of foreign matter when first seen, there will, in addition to the signs mentioned above, be some degree of respiratory distress with clinical signs of pulmonary collapse. It is this stage that is very frequently managed wrongly. What is needed is again immediately to put the patient in a posture which will enable gravity to protect him from further inhalation of foreign matter, and active tracheal and bronchial toilet by suction, either *via* a sterile rubber catheter passed into the trachea or by bronchoscopy. Too often one sees such patients being nursed in the sitting position, which seems to be the traditional position in which to place the dyspnoeic patient. This can only lead to further entry of foreign matter into the trachea and bronchi.

Oxygen administration is often indicated in these cases and is of value if posture and active suction and physiotherapy fail to clear obstructed bronchi or where simple mechanical obstruction has already progressed to bronchopneumonia.

III. *Patients with impairment of both ventilatory and protective components*

In this group, it is obvious that both active ventilation of the lungs and protection of the respiratory tract are necessary —in fact a combination of the treatments outlined above. The means of providing protection, however, may need to be modified to prevent the active ventilation from forcing secretions into the respiratory tract against gravity in spite of posture.

MANAGEMENT

It is the active ventilatory assistance required in groups I and III above that is most frequently neglected in respiratory emergencies, and often no attempt is made to supply this assistance until complete cessation of all respiratory efforts on the part of the patient. By this time myocardial failure is imminent and anoxia may have been present for a sufficient length of time to cause permanent damage to the central nervous system.

In the main, patients in category I require active ventilation, and this should be started too early rather than too late. A rapid clinical assessment of ventilatory adequacy can be made quite easily, without the use of spirometric apparatus,

by simply asking the patient to take as deep a breath as possible and count out loud for as long as possible. The average normal adult can count up to 40 or 50 with a single full breath. If this ability to count is found to be reduced to 20 or 30, active ventilatory assistance will most probably be necessary if a further degree of respiratory failure due simply

Fig. 1. The self-inflating Ruben resuscitator in use.

to retention of CO_2 is to be avoided. A count of below 20 indicates that assistance is already overdue and is therefore most urgently needed. Adequate ventilation should be maintained by whatever method is available and artificial respiration by Schafer's method or Eve's rocking method may be necessary in the first instance if paralysis is complete. Such methods however, soon become tiring, and a hand respirator or even an anaesthetic machine should be used if available or if paralysis is incomplete. Various pieces of equipment are obtainable for providing manual artificial

Fig. 2. The Oxford inflating bellows (left) and Kreiselmann resuscitator.

respiration, this being far more effective than the first-aid methods. Good examples of these are the Ruben resuscitator, the Kreiselmann resuscitator and the Oxford inflating bellows (Figs. 1 and 2). It does not matter what method is used, so long as it is efficient and sustained.

Patients in category II require no more than correct posture and tracheo-bronchial toilet as has been mentioned.

Patients in category III present a combined problem in that they require both protection and ventilation. They should be placed in a head-down position at once and maintained thus with the application of first-aid methods of artificial respiration until more adequate measures can be taken. As soon as possible, a cuffed endotracheal tube should be passed and the cuff inflated. This can very often be done without any anaesthesia or with topical anaesthesia alone. Once in position with the cuff inflated, such a tube at once isolates the trachea and bronchi from the mouth and pharynx. It provides excellent protection against the entry of foreign matter into the lungs and will enable a suction catheter to be passed. Active ventilation can be carried out *via* this tube by any method available, preferably an efficient manual inflator such as the Oxford bellows or Ruben resuscitator. Thus

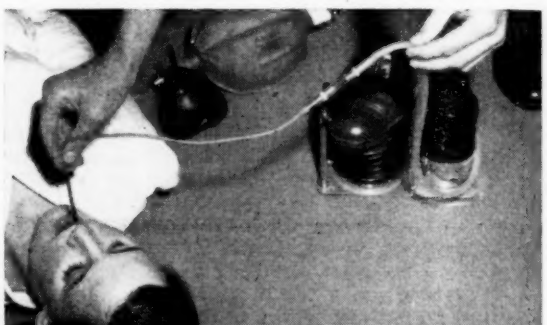

Fig. 3. Foot-operated portable suction machine in use.

protected and ventilated the patient can be safely transported to a centre where prolonged respiratory therapy can be carried out. If a cuffed endotracheal tube is used, it is advisable to deflate the cuff for 1 minute at intervals of 4-6 hours. This is in order to prevent ischaemic damage to the tracheal mucosa from pressure. Before releasing the cuff it is important to tip the patient head down and aspirate all saliva etc. from the pharynx (Fig. 3). Every effort should be made to reach a properly equipped centre for prolonged respiratory treatment within a period of 8 hours so that a cuffed tracheotomy tube may be substituted. If the journey is to be one of less than 100 miles then it may be made by road, but for greater distances it is far more practical to travel by air. During the journey ventilation still has to be maintained and the services of a trained anaesthetist should be obtained for this task if possible.

In the above, 3 clear-cut categories have been dealt with. It must be realized however that varying degrees of overlap will occur and that a patient in say, category I may rapidly or insidiously change to category III. Continued vigilance is therefore necessary on this score.

SUMMARY

1. The causes of respiratory failure, with or without, paralysis of the protective mechanisms of the pharynx and larynx, are discussed; together with the recognition of these conditions.

2. Stress is laid on the fact that accumulation of CO_2 is as dangerous as lack of oxygen, and that, without adequate ventilation of the lungs, neither can be properly corrected.

3. The emergency treatment of patients with these conditions, and their transport to a centre fully equipped for the necessary long-term management is outlined.

We wish to thank Mr. B. Todt for kindly taking the photographs.

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THE PREVENTION OF POST-OPERATIVE ULCER AFTER SURGERY FOR DUODENAL ULCER*

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Apart from the duodenal ulcer which is left *in situ* at the original operation for reasons of irresectibility, and apart from those ulcers which remain in short-circuiting operations (which residu-ulcers sometimes give rise to symptoms), we must admit at the very outset that secondary or post-operative, newly formed ulcers are peptic in origin.

Whatever the contributary factors in peptic ulceration, whether they be mechanical, or vasomotor, or other, the primary factors are the gastric juices. Notwithstanding the fact that peptic ulcer has been described in a so-called anacid stomach, we are still obliged to accept that our procedure has failed in these cases because we have not adequately suppressed the erosive acid effects; that is, *that our surgical attack has been inadequate*. Let me briefly review various surgical procedures as I see them:

1. *Gastro-Enterostomy*

There may be amongst you those who feel with Farquharson[1] 'that the incidence of post-gastrectomy symptoms varies directly with the amount of stomach removed', and therefore give preference to gastro-enterostomy for the treatment of a surgical duodenal ulcer. I admit that good results have been obtained with gastro-enterostomy, but I have never adopted it except for the really poor-risk, old-age case who has an organic pyloric stenosis.

I think it is universally admitted that:

(*a*) The bulk of stomal ulcers must be debited to an original gastro-enterostomy. I need hardly refer here to statistical proof (Glenn[2]).

(*b*) We find, when we turn to the experience of the largest clinics, that that most distressing sequela to ulcer surgery, a gastro-jejuno-colic fistula, has been preceded in almost 80% of cases by a *retro-colic* gastro-enterostomy (Lowdon,[3] Marshall[4]).

(*c*) A further drawback is that the passage of time does not afford the security one desires. After a resection stomal ulcers are prone to recur within a relatively shorter period, whereas over 50% of stomal ulcers following gastro-enterostomy occurred in the 5-10th post-operative year, i.e., *a late period* (Thompson[5]).

More and more stomal and jejunal (quite aptly called 'man-made') ulcers are discovered the longer the follow-up period after gastro-enterostomy: *a forcible reminder that only long-term assessments* will offer us *a true reflection of the value of any operation* for duodenal ulcer.

2. *Vagotomy*

Vagotomy as such has been replaced by vagotomy plus gastro-enterostomy owing to disturbances in motility caused by vagotomy without the latter. Whether this addition to gastro-enterostomy will restore it to a competitive place in surgery for duodenal ulcer time will have to show.

I have thus far used vagotomy only in re-resections for stomal ulcer, for it seems *that these cases demand every possible additional safeguard* against the erosive effects by hydrochloric acid. Vagotomy may also be advisable in addition to resection in the young person where surgery is indicated.

3. *Resection*

The term *partial gastrectomy* has turned out to be an unfortunate one, for it denotes removal of anything from a small segment to a large portion of the stomach, *and, therefore interpretation of its results becomes most confusing*. This was effectively shown by Capper's[6] survey of a large series of cases. He drew a comparison between cases with less than 70% and those with more than 70% of the stomach resected. In the Billroth II operation the difference for stomal ulcer was insignificant. The continual flush of bile over the stoma in this type of operation gives an added protection which is lacking in the Billroth I type. In Billroth I the recurrence rate was 8·2% for the smaller resections, and 0·9% for the large resections.

Then again, the term 'subtotal resection' conveys a *too total* conception. The history of gastric resections started with a truly partial sacrifice of this viscus. As time went on and post-operative ulcers were still developing, more stomach was extirpated. The portion of the stomach resected increased from one-half to two-thirds, and then to three-quarters and more than three-quarters. The less of the stomach left, the more frequently the patient presented with other post-operative morbidity: dumping, and inability to regain body-weight. It is at this cross-road in the problem of resection that we have arrived today!

Because of all this confusion I want to submit that the time has arrived when we are compelled to come to some definite agreement regarding the extent of our resection. It is useless persisting in talking about 'partial gastrectomy' when we really do not know what exactly we are talking about!

Unfortunately there are no precise anatomical points to select as our landmarks. Attempts at exact measurement (by the use of a polar planimeter) have for practical reasons not replaced the surgeon's guess.

May I emphasize that it is folly to gauge the extent of a resection by checking on how large a portion of stomach is removed. One must assess the extent *by what remains*. The fundic portion is the least visible part of the stomach, and one must actually see this portion clearly. This is best accomplished by bringing it down together with the spleen

* Paper read at the First Congress of the South African Association of Surgeons, Cape, Town April 1958.

into full view, placing a large abdominal swab above these structures, and *then* choosing the point of transection on the greater curvature.

What I leave, after my resection, is slightly less than the size of my fist for Billroth I, and no more than the size of my fist for Billroth II (Fig. 1). Since adopting this policy I have felt less uncertain of what I actually should attain. I now have something definite to designate an 'adequate resection'.

Distally there is no uncertainty. It is plain to all of us that we *must* go beyond the pylorus. There is sufficient evidence in the literature to show that failure to remove this section of stomach completely contributes to maintained delivery of the hormone gastrin, and that such antral remnants definitely increase the incidence of stomal ulceration (Marshall[7]).

Removal of the duodenal ulcer itself is a moot point. My aim is to remove it wherever possible. There are authors (Strauss,[8] Glenn,[2] Thompson[5]) whose impression it is that stomal ulceration is more frequent after exclusion than after removal of the duodenal ulcer. But, more important is the possibility of haemorrhage from an excluded ulcer. Marshall,[4] in reviewing the cause of death in 51 fatalities following resections, quotes 2, that is 4% of deaths, as a result of such haemorrhage.

I try to get distal to the ulcer in all cases, especially in a bleeding ulcer. I try to obtain a finger-breadth of intact posterior duodenal wall by inserting my left index finger into the lumen and dissecting on it in order to have sufficient margin for a Billroth I anastomosis, to which I am at present still inclined to give preference. The incidence of stomal ulceration after an *adequate* Billroth I resection must still be established. Less post-operative morbidity, and a shorter period of sequelae seems to be in its favour. The difficulties of dissection of the duodenal stump are the numerous small bleeders; it is a minute step-by-step dissection. The danger, to my mind, is not so much the common bile-duct (so often stressed) as the duct of Santorini which, in a number of cases, may form the main pancreatic duct.

BILLROTH I AND II OPERATIONS

Technically 3 basic criteria must be satisfied for both the Billroth I and II operations:

1. No tension. I invariably commence mobilization high up along the greater curvature just below the short gastric vessels which run to the superior pole of the spleen. This solves practically all my problems on this point (Fig. 2).

2. All knots and suture-material of the posterior sero-serous layer, even using catgut as I invariably do, should be buried by the overhanging muco-mucus layer. Material projecting into the lumen sometimes forms a predisposition to ulceration. I have seen these stomal ulcers with a silk suture projecting from the centre.

3. No damaged tissue should be employed at the stoma. Clamped or crushed tissue should be excised, and viable epithelium applied to viable epithelium.

Any duodenal stump not suitable for a safe Billroth I anastomosis forces the surgeon to the Billroth II procedure. 'Blow-out' of the duodenal stump as a 20% contribution to fatalities must be borne in mind in Billroth II. In performing a Billroth II anastomosis I never employ a retro-colic anastomosis. I fully realize that tissue susceptibility increases with the distance from the pylorus, but one's chances of a gastro-jejuno-colic fistula are ever so much less, and an eventual re-resection for stomal ulcer is so much easier, after an ante-colic anastomosis.

Further, I attach great importance to the location of the completed stoma in Billroth II. By drawing the transverse colon and omentum through well to the right, one firstly avoids the ante-colic jejunal loop becoming adherent to the laparotomy scar and causing trouble; and secondly one directs the loop in an even horse-shoe curve carrying the efferent limb to the left para-colic gutter (Fig. 3) thus minimizing torsion, internal herniation and similar mishaps which may follow the Billroth II anastomosis. I utilize the Finsterer type, right to left anastomosis, making the stoma no larger than for the Billroth I.

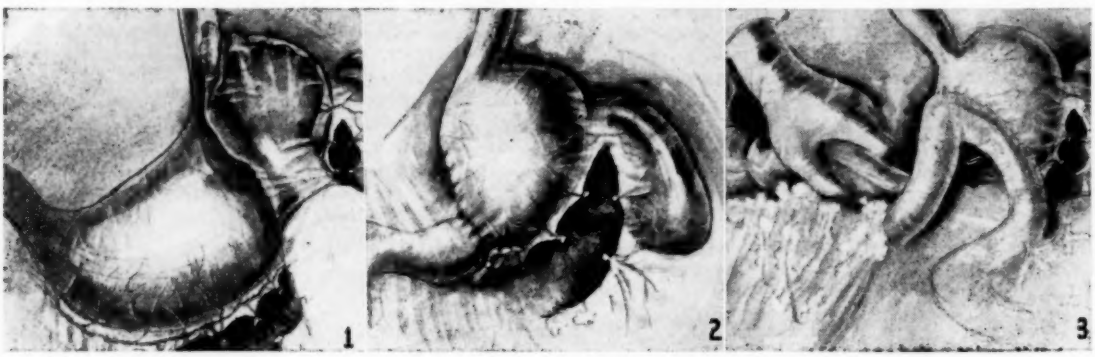
All cases stay on probanthin therapy for 3 weeks post-operatively, and on mashed, non-stimulating food for 3 months.

SUMMARY

A. An inadequate Billroth I resection increases the percentage of stomal ulcer.

B. A too drastic Billroth II resection increases and prolongs post-operative morbidity.

Finally, what about a less radical (40%) resection plus vagotomy? Again, time will have to show. Edwards[9] has reported some 300 cases of Billroth I and II with no stomal ulcer. But the finding that 26% of cases of gastro-entero-



Figs. 1, 2, and 3. Stages in Billroth operations.

stomy plus vagotomy show a return of acid to pre-operative levels after 4 years (Mayo clinic[10]) makes one hesitant. Long-term results should show the way.

For the treatment of recurrent ulcer I perform a thorough re-resection plus vagotomy.

I am greatly indebted to Dr. I. Henkel for the sketches.

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VALUE AND RELIABILITY OF THE BACITRACIN SCREEN TEST FOR IDENTIFYING LANCEFIELD GROUP-A HAEMOLYTIC STREPTOCOCCI

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S.haemolyticus is one of the most important pathogens of man and many of the bacteriological examinations carried out in a routine diagnostic laboratory are concerned with the identification of this organism.

In examining serial throat swabs of children suffering from scarlet fever or rheumatic fever, it was decided to evaluate Maxted's 'bacitracin screen test' as a means of identifying pathogenic haemolytic streptococci. A Lancefield grouping was carried out in parallel as a control on the bacitracin sensitivity results.

The test orginated with Maxted's observation that the growth of some haemolytic streptococci was inhibited by an adjacent growth of *B.licheniformis*, the inhibiting substance of which is almost identical with the antibiotic bacitracin.

In his test series Maxted showed that group-A streptococci were sensitive to bacitracin while other groups were resistant. He grouped or typed all strains in parallel and stated that as compared with Lancefield grouping, 1·7% appeared to be false sensitive strains and 2·5% to be false resistant.

The present investigation involved screening 520 strains of streptococci obtained from specimens of sputa and pus, nose, eye, vaginal and rectal swabs, and blood cultures.

METHOD

In order to use the least time-consuming technique all swabs were planted on horse-blood agar and the strains grown as surface colonies. It was thus possible to pick off colonies quickly for further investigation. The following morning all suspicious colonies were transferred either directly to fresh-blood plates or subcultured into broth or serum broth. In a few hours sufficient growth had developed in the broth tubes to transfer to a blood-agar plate, which becomes the diagnostic or 'sensitivity' plate carrying 4 strains. One half of each segment of the plate, on which were placed the discs soaked in a bacitracin solution of 5 units per ml., was inoculated heavily, and the other half was inoculated with 1 or 2 light strokes only. This facilitates recognition of contamination. It was essential to pick off at least 2 colonies from each specimen as experience showed that 2 almost identical colonies could give different results with regard to their bacitracin sensitivity.

When a strain produced a doubtful haemolysis it was incubated anaerobically. This usually clarified the type of haemolysis. It occasionally happened that, on the diagnostic plate, a strain showing bacitracin sensitivity changed its haemolytic property, became alpha-haemolytic, and grew as a resistant strain into the area of inhibition. Four such strains were encountered. In one instance the apparent beta-haemolytic streptococcus was in reality alpha-haemolytic for, when apparent alpha- and beta-haemolytic colonies were picked from the same plate and incubated under an atmosphere of CO_2, both types of colony presented alpha-haemolysis which was maintained on subsequent subcultures. In the other 3 cases, however, incubation under CO_2 was followed by the production of pure beta-haemolysis, which also was maintained during 5 subcultures (this phenomenom is at present being investigated).

In carrying out the parallel grouping, group-A serum only was used; cross reactions could therefore not be excluded. The following results were obtained: With the Lancefield grouping 380 strains were identified as group A and 140 strains were not. Of 383 bacitracin-sensitive strains 8, i.e. 2·1%, were found not to belong to group A when tested by Maxted's modification of the Lancefield grouping, and of 137 bacitracin-resistant strains, 5 i.e. 3·5%, proved to be false resistants. These false results show a slightly higher percentage than those published by Maxted.

TABLE I. RESUME OF RESULTS

	No. of Specimens	*Lancefield A Pos. Bacitr. Sensitive**	*Lancefield A Neg. Bacitr. Sensitive*	*Lancefield A Neg. Bacitr. Resistant**	*Lancefield A Pos. Bacitr. Resistant*
Throat swabs	419	326	4	86	3
Sputa	52	19	1	32	0
Pus	36	26	3	5	2
Eye swabs	2	1	0	1	0
Vaginal swabs	6	1	0	5	0
Rectal swabs	3	1	0	2	0
Blood culture	2	1	0	1	0
Total	520	375	8	132	5

* These columns show identical results of the Lancefield grouping and bacitracin sensitivity

DISCUSSION

In requesting bacteriological examination of specimens from the upper respiratory tract, or from foci of infection elsewhere in the body, the clinician is anxious, not only to know the bacterial flora which is present, but also the exact pathogens and their sensitivity to antibiotics. In so far as haemolytic streptococci are concerned, definite tests for classification may be beyond the capacity of the small laboratory. Few small laboratories carry out definitive tests for soluble haemolysin, leucocidin, fibrinolysin or erythrogenic toxin. Even the serological grouping of haemolytic streptococci is seldom available in such a laboratory and, in the absence of facilities for carrying out Lancefield grouping, the laboratory may rely solely on the presence or absence of beta-haemolysis which Schottmueller, as long ago as 1903, suggested as a means of classifying this group of organisms. Haemolysis itself, however, is subject to variation according to the environmental conditions and, even with the greatest care, is sometimes an

unreliable criterion of classification. In addition beta-haemolysis is frequently, but unjustifiably, equated with pathogenicity.

Under these circumstances a simple, reliable test which can be carried out in any laboratory, and which parallels the serological identification of haemolytic streptococci of Lancefield group A with reasonable accuracy, is to be welcomed. Maxted's screening test, using bacitracin, appears to meet this need. It is of no value in so far as anaerobic streptococci are concerned because, as Colebrook has shown, few of them are haemolytic. It might also fail in the presence of haemolytic streptococci acquiring bacitracin resistance owing to therapy with bacitracin but, so far as we know, none of the cases reported in this series had been so treated.

SUMMARY

A report is given of an investigation for bacitracin sensitivity of 520 strains of beta-haemolytic streptococci. The recommended concentration of 5 units of bacitracin per mil. was used with the disc technique. All strains were planted as surface cultures and incubated aerobically. Of 383 bacitracin sensitive strains 8 (2·1%) were false sensitive and of 137 bacitracin resistant strains 5 (3·5%) were false resistant.

I wish to thank Mr. P. Roux, of the bacteriological department of the South African Institute for Medical Research, for the encouragement I received from him when discussing this paper, and Mr. R. Robinson of the same department for his technical advice.

REFERENCE

Maxted, W. R. (1953): J. Clin. Path., 6, 224.

MENTAL ILLNESS IN THE UNION : DEBATE IN PARLIAMENT

BY OUR PARLIAMENTARY CORRESPONDENT

Nine members of the medical profession spoke in the two-day debate on the health portfolio in the House of Assembly on 9 and 10 September 1958. The debate covered a wide field. The chief concern was about three matters—the state of facilities for the treatment of patients suffering from mental illnesses; the need for coordination of health policy and services and the high cost of modern drugs and treatment. The Minister of Health (Mr. M. D. C. de Wet Nel) was in almost complete agreement with all the speakers had to say on these matters.

The main critics of the existing facilities for the treatment of mental patients were Dr. E. L. Fisher, M.P. for Rosettenville, and Dr. A. Radford, M. P. for Durban-Central. Dr. Fisher said the position in South Africa had followed the pattern throughout the world. Of all the diseases that beset the population the most widespread were those associated with mental illness—10 percent of the population were suffering from some or other mental ailment yet less was spent on the treatment and study of mental diseases than any other disease. Ten times as much was spent on tuberculosis and 50 times as much on cancer.

The time had come for a courageous Minister of Health to set a pattern for the rest of the world by setting aside large sums in South Africa for an investigation of the treatment, housing and nursing of mental patients.

THE STAFF PROBLEM

First of all the right incentive would have to be provided to attract sufficient staff of the right sort to this type of work. The conditions of employment must be made more attractive, they must be given opportunities for study and research and they must be guaranteed employment after qualifying. It was unfortunate that medical officers often were transferred from one mental hospital to another on promotion. This disturbed not only their own home life but also the continuity of treatment of the patients. Medical officers working in mental hospitals would have to be assured of promotion without having to move to other hospitals.

Dr. Fisher produced a petition signed by 1,000 friends and relatives of patients in Valkenburg Hospital, Cape Town, complaining that the conditions of service for the staff there were not such as to attract the best people.

To alleviate the shortage of accommodation in mental hospitals and to obviate the necessity for many patients to be taken hundreds of miles from their families for treatment, Dr. Fisher suggested that the provincial hospitals be asked to make beds available (for the treatment of mental patients who were not dangerous) under the supervision of the Commissioner for Mental Hygiene.

Provision could also be made in the provincial hospitals for the accommodation of immediate acute cases pending their removal to a larger hospital and for the treatment of chronic patients in an out-patients department. These chronic patients could return to their homes every day to be cared for by their own families or visiting nursing aides. He felt sure that all psychiatrists in the districts concerned would be happy to cooperate in schemes of this nature.

Dr. Radford said the responsibility of the State towards mental patients was a peculiar and a grave one because the inmates of mental hospitals were deprived of their liberty without their consent. It was the duty of the State to see that they were well cared for and where possible returned to the community. But conditions in this country were such that this was well-nigh impossible. A few gallant doctors were doing their best and returning some people but on the whole they were frustrated.

ACCOMMODATION PROBLEM

The problem of accommodation had become so acute that last year there were 2,213 more patients than beds in mental institutions. Over-crowding had a deleterious effect on the mentality of patients. It made it impossible to separate the noisy from the tranquil; the dirty from the clean; the maniacal from the harmless. It placed a tremendous obstacle in the way of recovery—one which could not be overcome by any conceivable method of therapy.

Beds were only a few inches apart and in some cases were in the centre of the room as well leaving no open passage. It was not surprising in view of the excitable patients occupying these dormitories, that recently a patient had murdered a fellow-patient in the next bed before the attendants could intervene. Non-Europeans in some dormitories lay shoulder to shoulder and feet to shoulder as well, making a solid layer of humanity with scarcely room to put a foot between them.

Fort Napier Hospital was built as a barracks in 1861. It was a wood and iron building propped up in places by gum poles to stop it from blowing over. There was no heating and fires could not be lit there. Among the patients were aged European females. The place had been condemned in 1936 but was still being used as a mental hospital.

THE MINISTER'S REPLY

The Minister admitted frankly that he was unhappy about the situation. His predecessor had also been concerned about it and had appointed a commission whose report had just been made available. He was studying that report and in the meantime a start had been made on the vast backlog that had to be overtaken. There was to be a big mental hospital at Stikland which would cost £2,500,000 and would provide for 1,800 patients. Provision would be made for the establishment of accommodation for a further 2,592 mental patients when that hospital was completed. There was an amount in the estimates for the rebuilding of Fort Napier where the conditions were truly shocking.

Dr. Fisher's suggestion about the treatment of less dangerous cases in provincial hospitals had already been advanced by some of the provinces themselves and discussions were being held with provincial authorities to see in how far the scheme was practicable.

Replying to points raised by other speakers Mr. de Wet Nel agreed that the National Health Council had become unwieldy and ineffective—in fact it had long ago ceased to function—and that there was need for a revision of health policy and services with a view to better coordination. He had been holding discussions for some time already in this connection. There was

the eight-member coordinating council for hospitals and medical services which was doing excellent work but he was not satisfied. What he envisaged was a central health council composed of experts who could actively advise and assist the Government. He would do everything in his power to achieve this ideal.

The Minister said he was as disturbed as the doctors who spoke were about the high costs of drugs and medical treatment. He was strongly considering appointing a commission to go into the matter, especially the question of expensive medicines, to see what could be done to bring some measure of relief.

MENTAL HEALTH CAMPAIGN

Tara and Sterkfontein Hospitals are organizing a Mental Health Campaign from 1 to 18 October 1958. The purpose of this campaign is to inform the medical profession, ancillary services, nurses and the general public on matters to do with mental health and mental illness. Emphasis during the campaign will be laid on the fact that present-day psychiatric treatment involves more than just dealing with established mental illness. It also involves the prevention of serious emotional disorder or mental breakdown and the raising of the standard of mental health generally.

Amongst events to take place during this campaign, many are specifically for medical practitioners: The Southern Transvaal Branch of the Medical Association of South Africa has convened a special meeting of medical practitioners. This meeting will be held under the auspices of the Sub-group of Neurologists, Psychiatrists and Neuro-surgeons.

A study day for medical practitioners will be held at Sterkfontein Hospital, Krugersdorp, under the auspices of the Medical Graduates Association, on Thursday 9 October 1958. The subject of the study day will be 'Recent Advances in Psychiatric Treatment'. A most informative and interesting programme of talks and demonstrations will be arranged. Doctors wishing to have more details about the study day, should telephone the Secretary, Medical Graduates Association, 22-3529.

Other events in which medical practitioners may be interested are:

1. *Mental Health Display.* This is a pictorial exhibition illustrating facts of mental health and mental illness. It will be opened at 11.30 a.m. on Wednesday 1 October at Tara Hospital by Dr. H. J. Hugo, Director of Hospital Services, Transvaal Provincial Administration. It will remain open at Tara Hospital until 9 October and may be seen there at the following times: Wednesday 1 October 12 noon—9 p.m.; Thursday 2 October 9 a.m.—9 p.m.; Saturday 4 October 9 a.m.—9 p.m.; Monday 6 October 9 a.m.—9 p.m.; Wednesday 8 October 9 a.m.—9 p.m.

2. The public is invited to visit Tara and Sterkfontein Hospitals. Visitors will be shown around both hospitals, and an explanation of the work that is done will be given.

Tara Hospital. Conducted tours for visitors are arranged for Saturday 4 October and Wednesday 8 October.

Sterkfontein Hospital. Conducted tours for visitors are arranged for Thursday 2 October and Saturday 11 October. Full details of times and transport will be given on enquiry at the hospital concerned.

3. *Mental Health Film Shows* will be held at the Darragh Hall, Plein Street, Johannesburg at 8.15 p.m. on 1, 3 and 8 October.

In addition, an all-day conference on a topic of national importance entitled 'Workshop on Psychopathy' has been arranged at Tara Hospital. The problem of the psychopath concerns many organizations and professions, i.e. psychiatrists, the law, social welfare, etc. and a multiprofessional group with representatives drawn from the highest level will discuss common problems. This conference will be by invitation only.

THE EFFECTS OF NUCLEAR RADIATION: WORLD MEDICAL ASSOCIATION REPORT

The General Assembly of The World Medical Association was the first group of doctors to consider the biological effects of nuclear radiation after the issuance of the report of the United Nations Scientific Committee.

At the request of many of its 55 member medical associations, The World Medical Association initiated a campaign designed to keep the medical profession in every country fully informed on the effects of nuclear radiation. One of its first actions in this campaign was in securing Dr. Louis M. Orr of Orlando, Florida (USA), consultant to the Institute of Nuclear Studies, Oak Ridge, Tennessee, to address the 12th General Assembly of the Association on the subject *The Biological Effects of Nuclear Radiation.*

Dr. Orr reported: 'It is known that radiation can be dangerous but *fire* can be dangerous also. It is a matter of control. It is a fact that for centuries mankind has lived with an amount of radiation some 30 times greater than the fallout to this date from nuclear tests. It is also a fact that modern man has received a great deal more radiation over his entire body from X-ray and fluoroscopic examination than from nuclear fallout . . . In the medical profession it is known that radiation is an effective treatment for certain diseases and a necessary accompaniment of important diagnostic procedures. It cannot be discontinued nor should it be indicted indiscriminately. It is necessary to evaluate potential *dangers* against possible benefits'.

Dr. Orr discussed the hazards of radiation fallout in general, including the genetic effects of strontium 90 and compared the amount of radiation to which man has been subjected from natural sources to that from tests, and what would be the probable effects if tests were continued either at the present rate or at the highest rate of a few years ago. He noted that 'the 30-year dose to the gonads received by the average person in the USA is estimated to be (*a*) from background radiation—about 4·3 roentgens, (*b*) from X-ray and fluoroscopy—about 3 roentgens, (*c*) from nuclear weapons testing if continued at the rate of the last 5 years—probably only 0·1 of one roentgen. If nuclear testing were continued at the rate of the 2 most active years the possible exposure of 30 years would be only about 0·2 of one roentgen'.

Dr. Orr warned governments making nuclear tests to take every possible precaution. He was of the opinion that while there should be no indiscriminate testing of atomic devices, especially of thermonuclear weapons, neither should there be discontinuance of properly controlled testing that could contribute to knowledge that would be beneficial to all people in the fields of medicine, mining, chemistry, power generation and agriculture. He stated that some risk was justified, though every precaution should be taken to minimize that risk.

Information obtained by the WMA.

The doctors of the world have become so concerned about this whole problem, and the hysteria which has developed chiefly from ignorance, that they asked The World Medical Association to obtain information which could be distributed to the medical profession and through it to the people of the world.

A year ago, the United Nations informed The World Medical Association that it could not supply it with any advance information on this subject. Hence, The World Medical Association turned to areas which had information and were willing to release it for the mental and physical well-being of mankind. Dr. Orr was selected to speak on the subject of biological effects of nuclear radiation before the 12th General Assembly of The World Medical Association because of his consultant status at the Oak Ridge Institute and because he came from a country where the latest research information could be freely obtained.

The World Medical Association notes with justifiable pride that it received information prepared before the issuance of the Report of the United Nations Scientific Committee and without its contents being available to the speaker and that the facts it received are not controverted by the UN Report, which states that:

'Some hazards are implicit in almost all technological advances,' but radiation exposure in X-ray diagnosis and treatment, as well as in research and industry, are 'for the benefit of mankind and can be controlled'. In this respect the Committee drew a sharp distinction between hazards undertaken voluntarily and hazards imposed on all the peoples of the world without their consent.

However, The World Medical Association is of the opinion that there should be closer cooperation between international organi-

zations having the health of the people of the world as one of their objectives. The public is gravely concerned about events which may effect their health and longevity. They turn to their doctors for information on these subjects. The doctors must be kept informed of the most advanced scientific research findings in order to provide their patients with the true facts. The World Medical Association has pledged itself to continue its activity in supplying the information that the doctors of the world need 'to assist all peoples of the world to attain the highest possible level of health,' and 'to promote world peace'.

THIRD CONGRESS OF THE SOUTH AFRICAN PAEDIATRIC ASSOCIATION, PRETORIA, 9-11 OCTOBER 1958

The 3rd congress of the S.A. Paediatric Association will be held at the Pretoria Hospital administrative block, on October 9–11. A number of papers in the programme hold interest for practitioners not engaged in the practice of paediatrics. General practitioners in particular, will find much material of value, and it is hoped that as many as are interested will join in the congress. A cordial invitation is extended to all practitioners, both general and specialist, to attend the meetings and join in the discussions.

All the papers, with the exception of a few, will be of 20 minutes duration, to be followed by discussion. Provision has been made for social functions at night, such as a cocktail party and annual dinner as well as a mayoral luncheon, to which wives and partners are cordially invited.

During the day a Ladies' Entertainment Committee will provide transport and entertainment facilities for wives. Queries of any kind regarding sport, accommodation, etc., should be addressed to the Hon. Secretary, Dr. E. Fasser, 201 Medical Centre, Pretoria, (Telephone 3-7953).

PROGRAMME

Thursday 9 *October*, Administrative Building, Pretoria Hospital.

Morning Session

8.30—9 a.m.	Registration
9—10 a.m.	Dr. B. Epstein: Presidential address: Hospitalization of Children. A film on the subject will be shown. Tea Interval
10.30—11 a.m.	Dr. L. Shore: Pre-asthma
11—11.30 a.m.	Dr. P. Lanzowsky: Iron Deficiency Anaemia amongst Pre-school-going Children in the Cape Peninsula.
11.30—12.30	Dr. James Gear (Invitation Lecture): Recent Advances in Virology.
12.45—1.45 p.m.	Mayoral Luncheon, Fountains Valley.

Afternoon Session

2—2.30 p.m.	Dr. M. Bowie: The Hyperosmolarity Syndrome
2.30—3 p.m.	Dr. E. Kahn: Dehydration in Malnourished Children.
3—3.15 p.m.	Dr. Joan Scragg: Amoebic Liver Abscess in African Children. Tea Interval
3.30—5 p.m.	Annual General Meeting of the S.A. Paediatric Association.

Evening

A braaivleis for members and their wives or partners will be held at the residence of the President, Dr. B. Epstein. (Please note that the dates for the braaivleis and dinner have been interchanged).

Friday 10 *October* (Public Holiday), Lower Lecture Theatre, Clinical Building, Pretoria Hospital.

Morning Session

9—9.30 a.m.	Dr. M. H. Schnier: Neonatal Hyperbilirubinaemia.
9.30—10 a.m.	Dr. Joan Griffiths: Neonatal Oedema. Tea interval
10.30—11 a.m.	Prof. Findlay Ford: The Medical Aspect of Hypothermia.
11—11.30 a.m.	Dr. M. Winship: Whipworm Infestation in Indian and African Children.
11.30—12 p.m.	Dr. N. Mann: A Pilot Scheme for Bantu Infant Feeding.
12—12.45 p.m.	Dr. L. Jordan: The Artificial Kidney.

Afternoon Session

2—2.30 p.m.	Dr. H. Gordon: Craniostenosis in Infancy.
2.30—3 p.m.	Dr. B. Senior: A Disease in Search of a Name.
3—3.30 p.m.	Dr. Tito de Morais: Parasitological Aspects of African Children in Mozambique. Tea interval
4—5 p.m.	Dr. J. C. van der Spuy: Demonstration of Open Heart Surgery in Animal Subject at Veterinary Research Laboratories, Onderstepoort. Discussion of problems involved.

Evening

Dinner (formal dress) for members and wives or partners.

Saturday 11 *October*, Lower Lecture Theatre, Clinical Building, Pretoria Hospital.

Morning Session

8.30—10 a.m.	Joint session with Department of Medicine, University of Pretoria. Demonstration of Cases. Tea interval
10.30 a.m.	Paediatric Cardiology
11.30—12 p.m.	Dr. J. L. Brando: Patent Ductus Arteriosus, Mitral Stenosis in Infancy. Empirical Observations on Open Heart Surgery. Demonstration of Cases.

ASSOCIATION NEWS—CAPE WESTERN BRANCH : VERENIGINGNUUS—TAK WES-KAAPLAND

The following items of general interest were discussed at a meeting of the Branch Council of the Cape Western Branch held at Cape Town on 22 August 1958:

Die President het hulde gebring aan die nagedagtenis van wyle dr. J. S. du Toit wat sowel die Takraad en Federale Raad baie jare met onderskeiding gedien het, President was van die Vereniging en vereer is met die Vereniging se goue medalje. Dr. du Toit was gereeld teenwoordig op Takraad-vergaderings tot sy dood en sy ondervinding en leiding sal baie gemis word.

Federal Council. Dr. F. W. F. Purcell was elected by Branch Council to fill the vacancy on Federal Council caused by the death of Dr. J. S. du Toit.

Leipoldt Memorial Lecture. Prof. M. R. Drennan has consented to deliver the Leipoldt Memorial Lecture. The subject of his choice is 'The Role of Sex in Human Evolution'. He appears to have a very attractive new theory, and has spent some considerable time in research with regard to it. It has provisionally been decided to have this lecture in October. It will be an open lecture for members, their wives and interested friends.

Medical House (Pty.), Limited. Two Directors had to be elected to represent the Branch on the Board of Medical House (Pty.), Ltd.

in view of the resignation of Dr. A. W. S. Sichel and the death of Dr. du Toit. Drs. M. Helman and J. H. L. Shapiro were nominated and duly elected.

Treatment of patients able to afford private fees in closed hospitals. The *ad hoc* committee previously appointed by the Branch to investigate the above matter, reported that at round-table meetings with members of the staffs of the Karl Bremer and Groote Schuur Hospitals, the question of whether or not the 'closed' hospitals were competing unfairly with general practitioners had been discussed. The committee had in mind a scheme which they were anxious to submit to the Universities and the Provincial Administration to ease the tension which undoubtedly exists between those in private practice and 'closed' hospitals. This will be fully discussed at the next meeting of the Branch Council. Members of the Council will be circulated with an interim report on the discussions of the committee before the meeting.

Public Subscriptions for funds for overseas treatment. A resolution was submitted to the effect that the Branch should make representations to the Minister of Justice to instruct Magistrates not to grant permission for public collections for the purpose of sending patients overseas before referring the cases to the local Branch of the Medical Association. The purpose of this measure is to ensure that the public will not be called upon to donate large sums of money for overseas treatment of conditions that can very well be dealt with locally.

Free hospital treatment for wives and families of doctors on the honorary staff of provincial and state aided hospitals. The Cape Midland Branch appealed to this Branch for their support in requesting the Director of Hospital Services to allow free hospital treatment to wives and families of doctors on the honorary staff of Provincial and State-aided Hospitals. The Branch Council supported this view and it was agreed that the matter be referred to the Liaison Committee for consideration and that the Director of Hospital Services be advised of our opinion.

Memorial fund for Dr. J. S. Du Toit. Dr. A. W. S. Sichel advised that it had been felt that something should be done in memory of Dr. du Toit, and requested the approval and support of the Branch. The idea would be to raise sufficient funds to give an annual prize at the University of Cape Town for the best student in Ophthalmology, or, if a greater sum is obtained, a scholarship. The Council gave its support and approval to this scheme, the initial arrangements will be made by Drs. Sichel and J. K. de Kock.

IN MEMORIAM

T. A. FULLER, M.B., CH.B. (EDIN.) D.P.H., R.C.P.S. (EDIN.)

Dr. Jack Abelsohn of Cape Town, writes: The death occurred on 1 September 1958 at his home at Rondebosch, Cape Town, of Dr. T. A. Fuller, formerly Head of the Department of Anaesthetics at the Groote Schuur Hospital, and senior lecturer in anaesthetics to the University of Cape Town. He was 68.

Dr. T. A. Fuller
[*Photo: Cape Times*

His sudden passing came as a great shock to his numerous friends. Following a severe cardiac breakdown some two years ago, he made a very remarkable recovery and was actually doing a locum at the Groote Schuur Hospital at the time of his death.

Dr. Fuller 'uncle Tom', as he was affectionately known to his students and colleagues, graduated at Edinburgh University in 1914, and during World War I, he served in East Africa and Gallipoli. He was mentioned in despatches.

On his return to South Africa he practised for a while in the Gardens, and subsequently joined Dr. Sutton-Charnock in practice in the Rondebosch-Mowbray areas; for many years, while still a general practitioner, he acted as an honorary anaesthetist to the Rondebosch Cottage Hospital.

Early in 1939 he decided to specialize in anaesthetics, and proceeded to London for further study. At that time I had an appointment on the staff of the Anaesthetic Department at University College Hospital, and had the pleasure of meeting Dr. Fuller for the first time when he visited the hospital and introduced himself to me. I was very happy to be able to advise and guide him regarding some of his future activities and work in London.

He commenced practice as a specialist anaesthetist in Cape Town in 1939 and, subsequent to the death of Dr. Gordon-Forbes in 1941, he was appointed part-time senior lecturer in anaesthetics to the University of Cape Town. Early in 1948 he accepted this appointment in a full-time capacity, and in 1951 he was appointed Head of the newly formed Department of Anaesthetics at the Groote Schuur Hospital and was instrumental in laying the foundation of the very efficient department which he was able to hand over to his successor Dr. C. Jones, on his retirement in 1953.

He was well versed in his speciality, soundly conservative yet fully appreciative and cognisant of the rapid advances which were being made in the speciality during this period. He taught his students to administer a 'safe anaesthetic'.

Dr. Fuller had the honour of being elected the first chairman of the Cape Town Branch of the South African Society of Anaesthetists, and was a past President and an honorary life member of the South African Society of Anaesthetists. His advice and guidance were eagerly sought and always freely given.

He was an outstanding sportsman, having at one time been a Western Province tennis champion, and a former South African senior golf champion.

He is survived by his wife, son and a daughter, to whom our sincerest sympathies are extended in their bereavement. Sleep peacefully Uncle Tom—we shall often think of you—and miss you.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. P. G. J. Koornhof, Adjunk Mediese Gesondheidsbeampte, is aangestel vanaf 1 September 1958 as Hoof Mediese Gesondheidsbeampte van Bloemfontein.

* * *

Dr. L. Vogelpoel, of Cape Town, left on 11 September to attend the Third World Congress of Cardiology in Brussels where he will read a paper. Before returning to Cape Town on 26 October he will visit London for postgraduate studies.

* * *

Dr. and Mrs. V. Schrire, of Cape Town, left on 12 September for an extended visit to Brussels and the United States. In Brussels Dr. Schrire will read a paper at the Third World Congress of Cardiology on the electrocardiogram in beri-beri heart disease. During his stay in the USA, he will visit the main centres for the study of heart disease and cardiac surgery. Dr. Schrire has received an award from the Ella Lyman Cabot Trust and is being assisted by the Cape Provincial Administration.

* * *

Dr. W. v. d. M. Lambrechts, who is currently in the Department of Anaesthesia of the University of Cape Town at Groote Schuur Hospital, has been appointed Nuffield Dominion Clinical Assistant to the Nuffield Department of Anaesthetics at the University of Oxford. He will be leaving shortly to take up his 2-year tenure of this appointment.

Dr. W. v. d. M. Lambrechts, wat tans aan die Departement van Anestesie van die Universiteit van Kaapstad te Groote Schuur-Hospitaal verbonde is, is aangestel as ‚Nuffield Dominion Clinical Assistant' aan die Nuffield Departement van Anestesie van die Universiteit van Oxford. Hy sal binnekort vertrek om sy 2-jaar termyn onder hierdie aanstelling te aanvaar.

Johannesburg Group of Medical Women. The Annual General Meeting and official dinner of this group (S.A. Society of Medical Women, M.A.S.A.), will take place on Wednesday, 8 October 1958, at the Quality Inn, Ingram's Corner, Twist Street, Hillbrow, Johannesburg, at 6.45 p.m. All medical women are welcome.

Will those desirous of attending please communicate with Dr. Esther Neros, either in writing to P.O. Box 2616, Johannesburg, or by telephone, 22-4571.

* * *

Mr. Edmund L. Trott, M.R.C.O.G., has commenced practice on his own account as an Obstetrician and Gynaecologist at 810, Eagle Star House, West Street Durban. Telephones: rooms 6-7625, residence 88-6458, after hours 2-9326.

Dr. Edmund L. Trott, M.R.C.O.G., het begin praktiseer (op sy eie) as 'n Verloskundige en Ginekoloog te Eagle Star House, 810 Weststraat, Durban. Telefone: spreekkamers 6-7625, woning 88-6458, na ure 2-9326.

* * *

Cape Western Branch. The monthly meeting will take the form of a symposium on 'The Treatment of the late results of chronic pelvic infection in women' in the physiology lecture theatre, Medical School, Mowbray, on Friday 26 September 1958. The speakers will be Dr. D. P. de Villiers, Dr. J. O. E. Apthorp and Dr. P. F. M. du Toit.

Tak Wes-Kaapland. Die maandelikse vergadering van die tak sal die vorm van 'n samespreking aanneem oor „Die behandeling van die laat gevolge van kroniese infeksie van die bekken by vrouens' in die fisiologie-lesingsaal, Mediese Skool, Mowbray, op Vrydag 26 September 1958. Die sprekers sal wees dr. D. P. de Villiers, dr. J. O. E. Apthorp en dr. P. F. M. du Toit.

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Northern Areas Division, Cape Western Branch. The monthly meeting of the Division scheduled for Tuesday 23 September has been postponed until Tuesday 30 September. The details as given in the issue of the *Journal* of 13 September remain unchanged.

Afdeling Noordelike Gebiede, Tak Wes-Kaapland. Die maandelikse vergadering van die Afdeling wat op Dinsdag 23 September sou plaasvind, is uitgestel tot Dinsdag 30 September. Besonderhede van die vergadering soos aangegee in die uitgawe van die *Tydskrif* van 13 September, bly onveranderd.

* * *

Union of South Africa, Department of Health. Newsletter No. 35 *of* 1958. Notification of formidable epidemic diseases and poliomyelitis in the Union during the period 22-28 August.

Plague, Smallpox, Typhus Fever. Nil.

Poliomyelitis

	Eur.	*Nat.*	*Col.*	*As.*	*Total*
Transvaal	–	1	–	–	1
Cape Province ..	1	–	2	–	3
Orange Free State..	–	–	–	–	–
Natal	–	1	–	–	1
Totals	1	2	2	–	5

Local Authorities	*Eur.*	*Non-Eur.*
Transvaal:		
Rustenburg municipality	U	1
Cape Province:		
George municipality	U	1
Kenhardt divisional council ..	R	1
Walmer municipality	U	1
Natal:		
Eshowe district	R	1

R=Rural. U=Urban.

* * *

Unie van Suid-Afrika, Departement van Gesondheid. Nuusbrief nr. 36 *van* 1958. Opgawes van gedugte epidemiese siektes en poliomiëlitis in die Unie gedurende die tydperk 29 Augustus tot 4 September.

Pes, Pokkies, Tifuskoors. Geen.

Poliomiëlitis

	Bl.	*Nat.*	*Kl.*	*As.*	*Totaal*
Transvaal	1	1	–	–	2
Kaapprovinsie ..	–	–	1	–	1
Oranje-Vrystaat ..	–	–	–	–	–
Natal	1	1	–	–	2
Totaal ..	2	2	1	–	5

Plaaslike Besture	*Bl.*	*Nie-bl.*
Transvaal:		
Brakpan-munisipaliteit	S	1
Pietersburg-distrik	P	1
Kaapprovinsie:		
Kaapstad-munisipaliteit	S	1
Natal:		
Durban-munisipaliteit	S	1
Eshowe-distrik	P	1

P=Plattelands. S=Stedelik.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

ELASTOPLAST FIRST-AID CREAM

Smith and Nephew, in introducing a new first-aid cream containing domiphen bromide, supply the following information: Elastoplast First-Aid Cream contains domiphen bromide (dodecyl-dimethyl-2-phenoxyethyl-ammonium bromide), a modern antiseptic of the Q.A.C. type. It was selected after extensive research as the most effective general antiseptic available today. It rapidly kills germs and thereby prevents infection of injuries, thus permitting rapid healing. This antiseptic is an improvement upon older types because of its wide range of effectiveness against all the common micro-organisms and also because it retains its activity in the presence of pus and blood.

In addition to its first-aid use, the cream may be used as an antiseptic glove by sick room attendents, doctors, nurses etc. who may have to handle infected material. Smeared over the hands and rubbed in, the cream disappears, but leaves an effective antiseptic film over the hands as a protection against infection.

Elastoplast First Aid Cream is available in attractive collapsible tubes and doctors are invited to send for samples and literature.

BOOKS RECEIVED : BOEKE ONTVANG

External Collimation Detection of Intracranial Neoplasia with Unstable Nuclides. By G. M. Shy, M.D., M.R.C.P., R. B. Bradley, B. S. and W. B. Matthews, Jr., B.S., with a chapter on *Scintillation Spectrometry* by J. E. Francis, B.S., P.R. Bell, Ph.D. and C. C. Harris, M.S. Pp. vii+144. Illustrations. 30s. net + 1s. 8d. Postage Abroad. Edinburgh and London: E. & S. Livingstone, Ltd. 1958.

The Interference Microscope in Biological Research. By Arthur J. Hale, M.B., Ch.B., Ph.D. Pp. xi+114. 95 Figures. 20s. net + 1s. 4d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

Advances in Electrocardiography. Edited by Charles E. Kossmann, B.S., M.D., Med.Sc.D., F.A.C.P. Pp. viii+280. Illustrations. $9.75. New York and London: Grune & Stratton, Inc. 1958.

The Principles and Practice of Medicine. A Text-book for Students and Doctors. 4th Edition. By Sir Stanley Davidson, B.A. Cantab., M.D., F.R.C.P.Ed., F.R.C.P. Lond., M.D. Oslo and the Staff of the Department of Medicine, University of Edinburgh and Associated Clinic Units. Pp. xi+1067. 73 Figures. vii Colour Plates. 35s. net + 2s. 9d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

Progress in Psychotherapy. Volume III. Techniques of Psychotherapy. Edited by Jules H. Masserman, M.D. and J. L. Moreno, M.D. Pp. x+324. $8.50. New York and London: Grune & Stratton, Inc. 1958.

Ciba Foundation Colloquia on Endocrinology. Volume 12. *Hormone Production in Endocrine Tumours.* Editors for the Ciba

Foundation: G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch. and Maeve O'Connor, B.A. Pp. xii+351. 58 Illustrations and Cumulative Index to Volumes 1-12. 48s. net. London: J. & A. Churchill Ltd. 1958.

Diagnostic Laboratory Hematology. 2nd Edition, Revised and Enlarged. By George E. Cartwright, M.D. Pp. xi+250. 33 Figures. $6.75. New York and London: Grune & Stratton, Inc. 1958.

Ophthalmic Plastic Surgery. 2nd Revised Edition. By Sidney A. Fox, M.S.(Ophth.), M.D., F.A.C.S. Pp. xii+324. 149 Figures. $15.00. New York and London: Grune & Stratton, Inc. 1958.

Atlas de Radiologie Clinique de La Presse Médicale. (Série là 100—1953-1958.) 1,764 Figures. Paris: Masson et Cie. 1958.

Milestones in Modern Surgery. By Alfred Hurwitz, M.D. and George A. Degensheim, M.D. Pp. xvii+520. Illustrations. $15.00. New York: Paul B. Hoeber, Inc. 1958.

The Reticulo-Endothelial System in Growth and Tumour Formation. By Roger Wyburn-Mason, M.A., M.D. (Cantab.), M.R.C.P. (London). Pp. 206. 79 Illustrations. 42s. net. London: Henry Kimpton. 1958.

Chirurgie Infantile D'Urgence. By Marcel Fèvre. Pp. 718. 201 Figures. Broché 7.000 fr. Cartonné toile 8.000 fr. Paris: Masson et Cie. 1958.

Histopathologie der Tumoren der Kiefer und der Mundhöhle. Von Prof. Dr. E. Langer. vii+167 Seiten. 212 Abbildungen. DM 49.50. Stuttgart: Georg Thieme Verlag. 1958.

Röntgenatomie der Neugeborenen- und Säuglingslunge. Von Priv.-Doz. Dr. med. habil. Z. Zsebök. 160 Seiten. 286 Teils farbige Abbildungen. DM. 75.—. Stuttgart: Georg Thieme Verlag. 1958.

A Text-book of X-ray Diagnosis. By British Authors in 4 Volumes. 3rd Edition. Edited by S. Cochrane Shanks, C.B.E., M.D., F.R.C.P., F.F.R. and Peter Kerley, C.V.O., C.B.E., M.D., F.R.C.P., F.F.R., D.M.R.E. Volume 3. *The Abdomen.* Pp. xvi+883. 802 Illustrations. £6 0s. 0d. net. London: H. K. Lewis & Co., Ltd. 1958.

Nederlands Handboek der Psychiatrie. Onder Leiding van Prof. Dr. J. J. G. Prick en Dr. H. G. van der Waals, met Medewerking van Prof. Dr. P. A. H. Baan, Dr. H. K. G. Bartstra, Prof. Dr. P. J. A. Calon, Prof. Dr. Th. Hart de Ruyter, Dr. L. N. J. Kamp en Dr. P. C. Kuiper. *I. Algemeen Gedeelte.* Pp. xii+484. f.29.50 gebonden. Arnhem: Van Loghum Slaterus. 1958.

Psychopharmacology—Pharmacologic Effects on Behaviour. Edited by Harry H. Pennes, M.D., D.Med. Sci. (Neurology), with 41 Participants. Pp. xiii+362. 35 Figures. $8.00. New York: Paul B. Hoeber, Inc. 1958.

Pediatric Surgery. By Orvar Swenson, M.D., F.A.C.S. Pp. xiii+740. 980 Illustrations. $20.00. New York: Appleton-Century-Crofts, Inc. 1958.

Begutachtung Peripherer Durchblutungsstörungen. Von Prof. Dr. H. W. Pässler und Dr. H. Berghaus. xii+272 Seiten. 68 Abbildungen in 147 Einzeldarstellungen. DM 45.00. Stuttgart: Georg Thieme Verlag. 1958.

Expert Committee on Poliomyelitis. Second Report. Technical Report Series No. 145. Pp. 83. Figures. 3s. 6d. Geneva: World Health Organization. 1958. Local Sales Agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria.

Joint FAO/WHO Expert Committee on Brucellosis. Third Report. Technical Report Series No. 148. Pp. 51. 3s. 6d. Also Published in French and Spanish. Geneva: World Health Organization. 1958. Local Sales Agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria.

Expert Committee on Water Fluoridation. First Report. Technical Report Series No. 146. Pp. 25. 1s. 9d. Also published in French and Spanish. Geneva: World Health Organization. 1958. Local Sales Agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria.

Excreta Disposal for Rural Areas and Small Communities. By E. G. Wagner and J. N. Lanoix. Pp. 187. 73 Figures. 25s. Geneva: World Health Organization. 1958. Local Sales Agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria.

International Standards for Drinking-Water. Pp. 152. 20s. Geneva: World Health Organization. 1958. Local Sales Agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria.

Insecticide Resistance in Arthropods. By A. W. A. Brown, Ph.D. Pp. 234. 29 Figures. 25s. French and Spanish Editions in Preparation. Geneva: World Health Organization. 1958. Local Sales Agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria.

The Incurable Wound and Further Narratives of Medical Detection. By B. Rouechè. Pp. 177. 15s. London: Victor Gollancz Ltd. 1958.

Income Tax and Company Law in South West Africa. By David Shrand, M.Com., A.S.A.A., C.A. (S.A.) and E. Zwarenstein, C.A. (S.A.) (Hons.). Pp. vii + 254. 37s. 6d. Cape Town: Legal & Financial Publishing Company. 1958.

What Every Taxpayer should Know about Income Tax. 3rd Edition. By David Shrand, M.Com., A.S.A.A., C.A. (S.A.). Pp. 81. 11s. Cape Town: Legal & Financial Publishing Company. 1958.

REVIEWS OF BOOKS : BOEKRESENSIES

MODERN TRENDS IN ENDOCRINOLOGY

Modern Trends in Endocrinology. Edited by H. Gardiner-Hill, M.D., F.R.C.P. Pp. ix + 298 + (13). 15 Figures. 73s. + 1s 7d. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1958.

This 'Modern trends' series has achieved considerable popularity and there are at least 15 other volumes, each of which deals with a particular aspect of medicine. The advantages of this type of publication is that the field which can be covered is wide and there is no obligation on the editor to aim at covering the whole subject. In fact, there is no apparent limit to the number of series of each branch of medicine which can be published as a number of second series (and even a third series) testifies.

This volume covers a limited field of endocrinology. Some of the chapters are very readable reviews, written in a style suitable for one who has no special knowledge of endocrinology. The thyroid gland, for example, receives considerable attention and the chapters are written in an eminently readable and concise manner. So too are the sections on steroid therapy in the treatment of rheumatic disorders, the chapter on adrenal function tests as well as that on carcinoid tumours and serotonin. Some of the other sections are not as easy to understand and will be of greater interest to the endocrinologist rather than to the general physician. But the editor has collected a very powerful team of workers each of whom is a specialist in his own field, and they have all written authoritative reviews of their own special topic. Often they have given their own views even if these were not in full agreement with the rest of the literature. With the speed of endocrinological advance being what it is today, it is not surprising that there is not unanimity of opinion in all fields. Frank discussion and the airing of differences can thus only do good. For these reasons this new member of a rapidly expanding family is ensured of a warm welcome and the second series is awaited with considerable interest.

C.M.

DISORDERS OF AGING

The Neurologic and Psychiatric Aspects of the Disorders of Aging. Proceedings of the Association for Research in Nervous and Mental Disease, December 9 and 10, 1955, New York, N.Y. Volume XXXV. Editors: J. E. Moore, M.D., H. H. Merritt, M.D. and R. J. Masselink, M.D. Pp. ix+307. 79 Illustrations. 17 Tables. 68s. London: Baillière, Tindall and Cox Ltd. 1956.

This book provides an authoritative statement on present-day ideas regarding neurological and psychological deviations of senescence and improved methods of research, through the medium of essays, discussions, questions and replies.

The material is predominantly factual, descriptive and almost devoid of speculation, nevertheless the fear that such a production would be pedantic and uninteresting is soon dispelled by the

lucid language, jargon-free style and stimulating interjections of the lecturers and listeners.

The need for research is stressed by the concept of life as consisting of a brief mentally immature youth and a long steadily deteriorating middle age, senescence and senility.

Trauma, infection, neoplasm and metabolic disturbances compete and cooperate to disable man in his youth and if he reaches 'The Top of Hill Seventy' unscathed, his survival is challenged by degenerative diseases. Nevertheless despondency is unjustified, for a great variety of means to retard and combat the ravages of old age are available, and further momentous discoveries seem according to the book just round the corner.

This is an exceptionally interesting and instructive book.

I.M.H.

PAINLESS CHILDBIRTH

Psychoprophylactic Preparation for Painless Childbirth. By Isidore Bonstein, M.D. Pp. 143. 14 Figures. 12s. 6d. London: William Heinemann Medical Books Ltd. 1958.

Dr. Bonstein has dedicated this short treatise to Dr. Lamaze of Paris 'who revealed to thousands of women a new and exhilirating approach to motherhood'. This 'psychoprophylactic' approach, based on Pavlov's famous conditioned reflex, results in the physical and psychical education of the pregnant woman during the last weeks of pregnancy.

To maintain, however, that this preparation of the pregnant woman is 'absolutely different from all other methods practised, with a view to making confinements painless', is hardly accurate if one has studied Grantly Dick Read. The continuous administration of oxygen during the second half of labour and delivery appears to be the only difference. Besides the simple explanation of the psychophysiology of the brain and pain in childbirth, the 'psychoprophylactic' preparation of the patient is briefly discussed in 8 short lectures dealing mainly with respiratory muscular relaxation, the mechanism of labour, and the functions of the brain. This makes for easy reading particularly for the busy practitioner and the lay person.

However, one cannot agree with such statements as 'the deeply engaged head is an obstetrical complication', or 'anterior or lateral displacements of the uterus are abnormal', and a 'good bandage' would easily correct this malposition.

However these are not very serious faults and one can recommend this simply written and easily readable manuscript to those interested in 'painless childbirth'.

L.R.

CORRESPONDENCE : BRIEWERUBRIEK

MEMBERSHIP OF BENEFIT SOCIETIES

To the Editor: Considering the dissatisfaction about the administration of our Association and the suggestion of raising the subscription fees, let the Association do something which will help to strengthen it.

There are people coming daily to consulting rooms, or even have the audacity to call the doctor to the residence, who are unknown to the doctor and who state that they are members of some Benefit or Aid Society, without producing any evidence whatsoever that they actually are members.

I will suggest that our Head Office urgently informs all the 'registered' Societies on their list that unless their members produce their cards, in accordance with the regulations, no attention will be given to them unless for cash—the fee demanded by the practitioner.

It must often happen that the practitioner attends to the patient in good faith and even refers the case to a specialist, only to find out that the person is not a member of any Society responsible for the fee and that he cannot even be traced, having given a false name and address, or having disappeared from the address at which he resided. I will suggest that all practitioners instruct their attendants to demand the card or the cash, just as I have done. I am certain that this will be of benefit to myself and that my suggestion will benefit all of my colleagues.

N. Finn

27 C.N.A. Building
East London
29 August 1958

J. S. DU TOIT MEMORIAL FUND

To the Editor: A feeling exists among a number of the colleagues and friends of the late Dr. J. S. du Toit that something should be done to perpetuate his memory in recognition of the long and valuable service he gave so willingly to the Medical Association and to medicine in general.

In particular he identified himself with the Benevolent Fund of the Association and saw it grow, largely by his own effort, from its infancy to its present healthy state.

A suggestion has been made that a Memorial Fund be established with the object of assisting the sons and daughters of deceased colleagues in their medical studies which they might have been able to undertake had the parent not died.

Under a Trust Fund set up for the purpose, educational grants could be made from time to time which at present hardly come within the scope of the Benevolent Fund, the main function of which is to render financial assistance to the necessitous dependants of deceased colleagues.

We therefore venture through the columes of the *Journal* to launch an appeal for contributions from our fellow members and others who would like to participate in this undertaking.

Cheques should be made payable to the 'J. S. du Toit Memorial Fund', P.O. Box 643, Cape Town, and will be acknowledged from time to time in the columns of the *Journal*.

A. Landau
J. D. Joubert
J. K. de Kock
J. P. Immelman
J. C. Coetzee
and A. W. Sichel

P.O. Box 643
Cape Town
4 September 1958

THE PSYCHIATRIC DAY HOSPITAL

To the Editor: Having worked at the Marlborough Day Hospital in London from September 1957 until January 1958, I was most interested in Dr. Gillis' article on the psychiatric day hospital. Although the facilities provided by the day-patient section of Tara Hospital appear to be very similar to those of the Marlborough Day Hospital, there is one crucial and fundamental difference that must be considered. Dr. J. Bierer and his entire therapeutic team place great emphasis on an invaluable extension of the usual day-hospital regime, which they term 'therapeutic social clubs.' These 'clubs' provide an indispensable adjunct to the formal therapeutic procedures of the Marlborough Day Hospital and in fact Dr. Bierer often mentioned that without this vital addendum, the usual activities of the day-hospital (individual and group psychotherapy, occupational therapy, electro-convulsive therapy, drug treatments, etc.) would lose half of their effectiveness.

While full details of the rationale and structure of these clubs are provided in *Therapeutic Social Clubs*, I should like to mention that a fairly wide choice of different clubs is available each week night and on Sunday afternoons. Club activities range from group discussions, group therapy and psychodrama, to informal social gatherings, dances, musical evenings, etc. The clubs are organized by psychiatrists or by clinical psychologists. Patients of the Maudsley and Marlborough Hospitals formed the main body of the clubs which I attended, with the addition of a few people undergoing private psychiatric treatment and some interested members of the public.

In my opinion, there is an urgent need for a similar service in South Africa.

Arnold A. Lazarus

41, Hillsborough Mansions
Cor. Pretoria & Klein Streets
Hillbrow, Johannesburg
11 September 1958

1. Gillis, L. S. (1958): S. Afr. Med. J., 32, 881.
2. Bierer, J., ed. (1948): *Therapeutic Social Clubs*. London: Lewis.